UNIVERSITY OF PENNSYLVANIA

A HEMATO-RESPIRATORY STUDY OF 101 CONSECUTIVE CASES OF STAMMERING

BY

MAX TRUMPER

A THESIS

IN PSYCHOLOGY

PRESENTED TO THE FACULTY OF THE GRADUATE SCHOOL IN
PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR
THE DEGREE OF DOCTOR OF PHILOSOPHY

PHILADELPHIA

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PREFACE.

STAMMERING being such an age-old problem, there has accumulated a vast amount of literature on the subject, much of which has little value. There are however, especially in recent years, earnest students of this subject who have made systematic and extensive studies. Among these the following have been very helpful to me in this thesis: Ssikorski of Russia, the Gutzmanns of Germany, Küssmaul of Germany, Makuen in this country, and the younger school in Austria and Germany represented by Stern, Hoepfner, Liebmann and preëminently Froeschels. Bluemel of Denver, Colorado has contributed an excellent two-volume work on stammering, Robbins of Boston has advanced our knowledge of cerebral congestion, Travis of Iowa City has done able research by means of his phono-photographic studies of the stammerer's voice.

The advances made during the last decade in the highly specialized studies of respiration have been indispensable to this investigation of stammering. The classical works of Henderson and Haggard of Yale, and of Douglas, Haldane and his co-workers at Oxford have been of inestimable value. The studies of Edsall, Means, Mudd and the late Peabody of Harvard have also aided greatly by their clinical data on respiration.

MAX TRUMPER.

APRIL 2, 1928.

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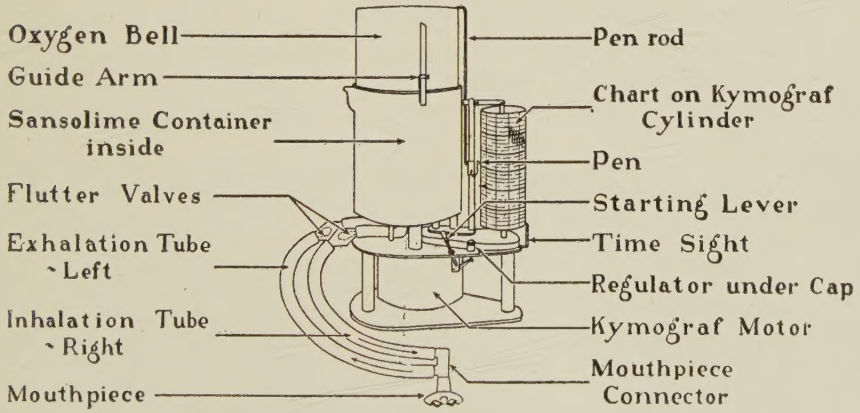
INTRODUCTION.

This investigation deals with 101 consecutive cases of stammering which appeared before the Speech Clinic of Professor Twitmyer.[1] It is a hemato-respiratory study of the cases taken just as they came to the Clinic and the term stammering will be used in its broadest sense. The literature is already overburdened with nomenclature, microscopic observations and telescopic deductions, therefore in this report, stammering is used as a collective term for various nervous forms of speech disorders but does not include the mechanical defects of speech. The subjects ranged in age from nine to thirty-two years, of whom 13 were females. Seventy-six of the cases were between fifteen and twenty-five years of age, 19 cases from nine to fifteen years, and 6 cases from twenty-five to thirty-two years.

I performed on every case the following tests: Basal Metabolic Rate, Volume per Breath, Respiratory Rate, Ventilation per Minute, Hemoglobin Estimations by the Sahli-Leitz and Newcomer Methods, Erythrocyte Count, Red Cell Volume, White Cell and Differential Blood Counts, and the Vital Capacity. A standard Sanborn graphic metabolism apparatus is pictured below:



[1] Graduate School of the University of Pennsylvania.

This was employed for all the tests. The kymograph is adjusted to a constant speed, half-minute intervals[1] being indicated by vertical lines on the chart. When the subject inhales oxygen from the apparatus, the tank drops and gives the pen a downward motion on the chart. This chart is calibrated in units of 100 cc. under standard conditions of temperature, pressure and aqueous vapor.[2] When the subject exhales into the apparatus[3] the tank rises and causes the pen to move upward. This alternate falling and rising of the oxygen tank is on a constantly decreasing level, the rate of descent indicating the volume of oxygen consumed. (See page 9.) Whenever it was desired to record also the subject's response to increasing amounts of carbon dioxide, the only change necessary was to remove the soda lime container before filling the oxygen tank. The subject's response then resembled the breathing seen during and after strenuous exercise. Case No. 25 on page 19 illustrates the normal response to this test.

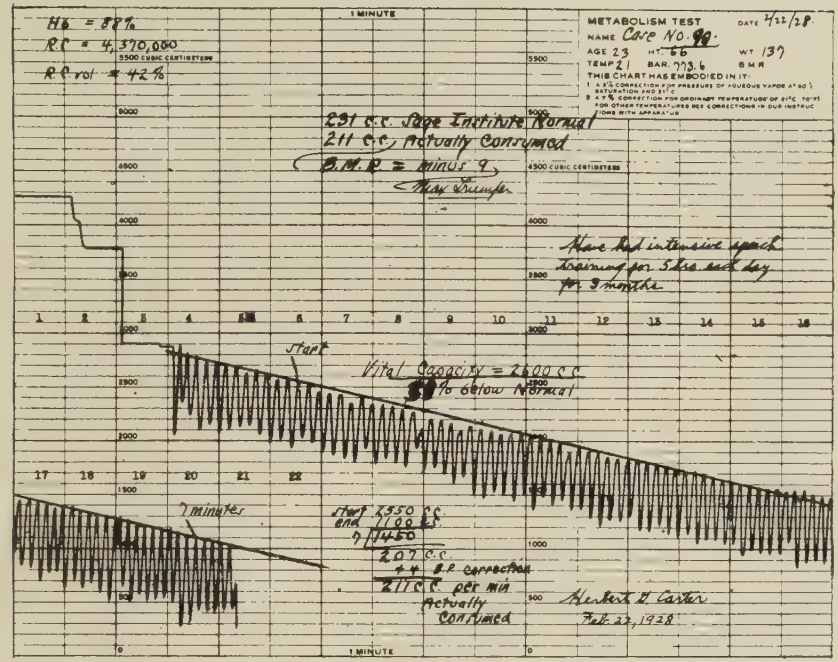
After making the tests and calculations on 12 cases, it seemed to me that there was a correlation of the type of breathing with the number of red corpuscles and hemoglobin content of the blood. To prevent this hypothesis from influencing my counting of the cells and estimating of the hemoglobin, I transferred this work to my assistant in the laboratory. The calculating of the volume per breath and the total ventilation per minute[4] was given to someone outside the laboratory. In this way the figures obtained were not affected by an unconscious desire on my part to see my hypothesis verified. Page 9 contains a sample chart with the calculations. The spaces at half-minute intervals are

[1] In every test, this speed was also timed with a stop-watch.

[2] This chart has embodied in it: a 2 per cent correction for pressure of aqueous vapor at 80 per cent saturation and at 21° C. Also a 7 per cent correction for ordinary temperature at 21° C.

[3] Carbon dioxide is absorbed by soda lime inside the oxygen tank.

[4] The total ventilation is the volume of air in liters breathed by an individual in one minute. Hendry, Carpenter and Emmes, Boston Med. and Surg. Jour., 1919, **181,** 285, 334, 368 have shown that the average carbon dioxide content of the expired air is 3.45 per cent and a correction of this amount should theoretically be added to the volume per breath because of the carbon dioxide absorbed by the soda lime inside the oxygen tank; but this was not necessary in this study.



Case No. 90.—H. G. Carter *Examined:* 2-22-28

Oxygen consumed per minute . .	211
Average respiratory rate per minute	11
Average ventilation per minute . .	6,591
Average volume per breath . . .	584
Dilution factor	31

211)6591(31 = dilution factor
633
261
211

Spaces No.	Respiratory rate.	Total ventilation.	Average volume per breath.
8–9	11	5365	488
11–12	10	5895	590
15–16	12	7350	612
18–19	12	7755	646
	4)45	4)26365	4)2336
	11¼	6591¼	584

Ventilation:

Spaces No. 8–9.	Spaces No. 11–12.	Spaces No. 15–16.	Spaces No. 18–19.
540	670	560	700
500	525	650	720
525	590	590	640
530	530	610	720
320	540	600	800
380	580	600	140
450	610	470	640
490	680	720	730
570	570	680	650
430	600	610	700
630	——	620	665
11)5365	10)5895	640	650
487–8	589½	12)7350	12)7755
		612½	646¼

numbered from 1 to 22, etc., to indicate from just what portions of the breathing curve the calculations are drawn.

Before presenting in detail the data obtained and my conclusions from these 101 cases, I shall deal first with the general aspects of stammering, second with the principles of respiration including the factors of vital capacity, so-called "dead space," tidal air, shallow breathing, and last with the subject of hematology.

STAMMERING.

One-half million people in the United States stammer. This is a conservative estimate and 80 per cent of these cases began before six years of age. The percentage of stammerers in special classes and schools for subnormal children has been found by Wallin (1915) and others to be several times greater than that in schools for normal children. Because of this fact, confusion has arisen and stammerers in general have suffered unjustly. The 101 cases here reported are not only normal but some have an intelligence quotient far above the average. I will cite one instance, Case No. 96, nearly nine years of age, the youngest of my group, who has an intelligence quotient of 150. His stammering is due to his abundant motivation. His speech difficulty has developed because the rapidity of his verbal thought exceeds his capacity for speech, that is, he thinks faster than he can speak.[1] Bluemel (1913) states that as far as intelligence is concerned, stammerers are normal persons. They probably vary between the two extremes as do persons of normal speech. Makuen (1914), who founded in 1896 in a poorly housed, congested district of Philadelphia, the first speech-defect clinic in this country,[2] reports that of his 1000 cases of stammering, 80 per cent were mentally alert. Therefore, these individuals are under greater phonatory and articulatory stress. Kenyon (1917) finds stammerers are usually of good mentality and, excepting for their stammering, are not different from other individuals of the same type. Robbins (1926) points out from his contact with 1000 cases that stammerers are usually of higher than average mentality. Twitmyer (1928) who has been treating stammerers for twenty-five years, says that the distribution and intellectual acquisitiveness of stammerers do not differ from non-stammerers.

[1] Ordinarily, one can speak about as rapidly as one can hear, some twenty changes a second.

[2] Philadelphia Polyclinic Hospital, now part of the Graduate Medical School of the University of Pennsylvania.

The mechanism of speech is a most complicated one, requiring exact mechanical inter-working of a number of muscle groups at a comparatively high rate of speed. Speech is undoubtedly one of the most, if not the most complicated of coördinated movements (Kickhefel 1924–1925). The three great components coöperating in its mechanical structure are: breathing, voice, and articulation. It will be impossible to consider the abundant anatomic and physiologic facts here, but mention should be made that each one of these three motor components has an activity more important than that of participating in vocal speech. The breathing muscles have to carry out the vital function of gas exchange. The articulatory muscles form part of the digestive tract. The voice apparatus serves as a protective device of the trachea and lungs against foreign bodies.

Because of the intricate mechanism of speech everyone will occasionally stumble on some word or syllable and in the same way Hoepfner (1911) compares incipient stammering to the attempts of a child learning to walk. During the first periods of the infantile development of speech, prior to the formation of words and sentences, there is a specific tendency toward the repetition of sounds and syllables. The child learning to walk stumbles and falls and the same thing happens in learning to talk. Walking is comparatively a simple coördination, yet if one had to consider carefully each step, walking would be very awkward. This stumbling in speech, unless there is a conspicuous reason for it, should be considered as a physiologic incident in little children, and in older children an occasional stammer from bashfulness or excitement should not cause concern. This emotional condition frequently gives rise to temporary stammering in children. The child attempts to describe something that has deeply impressed him. He gasps and gesticulates, but says nothing. But if the child is induced to calm himself before talking, he is able to talk slowly and fluently. But, if the child is made to repeat the words over which he has stumbled, then the parents or elders who do this are aggravating his difficulty. The stumbling then persists, increases, and may result in incipient stammering. What originally may have been simply an insufficient coördination between willing to speak and the finding of

words, now develops so that the child becomes conscious of difficult words and letters and gradually builds up a fear of speaking. His mind now becomes centered upon words rather than upon thought. The complicated process of speech coördination becomes less automatic. The memory of previous failures increases the fear of talking. Fear thus begins to be dominant. The stammerer's fear is very much like an aggravated form of stage-fright. The latter is an occasional condition experienced by the normal speaker. Stammering is a chronic though intermittent condition—intermittent because the stammerer usually is able to speak well in solitude. He speaks well also in concert with other people. Reproductive speech offers fewer difficulties than productive, while spontaneous speech is easier than reading. The stammerer rarely has difficulty in singing or swearing.

It is widely recognized that stammering follows physical shock as from a fall or blow and also follows psychic shock as from fright or fear. Shock, whether physical or psychical, checks or temporarily obstructs breathing, and since we talk and breathe alternately, the shock interferes with speech. This is apt to lead the patient to believe that he has lost his normal ability to speak. Whether shock constituted the entire cause, or whether such an individual was predisposed to stammer, is difficult to prove. But it is a fact that stammerers frequently give a history of shock.

Froeschels (1916) states that the early clonic or syllable repeating phase of stammering may arise from any of the following: (1) Deficient training in speech; (2) imitation; (3) divided attention or discrepancy between speech capacity and thought capacity; (4) beginning of a real aphasia developed during infectious diseases with high fever.

Froeschels has shown that the first stage in stammering is purely clonic, *i. e.*, the repetition of syllables. The next stage is that of a clonic-tonic condition, which finally becomes more or less pure tonus. The spasm-like or twitching movements accompanying stammering are both voluntary and involuntary; the latter constitute Froeschels' tonic phase characterized by squeezing of the speech organs. For instance, the patient at first repeats a sound, then articulates the latter every time with greater strength. He dwells too long and too impressively on the

respective position of the sounds. The frowning of the forehead, twitching of the nostrils, putting out of the tongue, protruding of the lips like a snout, are all involuntary. The secondary movements may appear in muscles remote from speaking. These are infinite in variety and of all degrees of intensity.[1] Often the individual may take refuge in embolophrasia, the inserting of irrelevant words to evade disaster. The stammerer becomes tied to certain words which are really accompanying movements having the character of sound. Others make endless attempts to get around what to them seems to be a difficult word by the use of synonyms or by a new formation of the sentence. Thus what may originally have been a simple and perhaps an incipient defect of speech has now matured into a serious nervous habit, sometimes complicated by psychical and personality abnormalities which may be concomitant phenomena or which, as is more probable, may be the results of the affliction. One who has not experienced this alarming predicament of suddenly being unable to complete what he is about to say cannot fully realize this affliction. It is more distressing than pain and its treatment has been undertaken by surgeons, physicians and psychologists.

Herewith follow the essential instructions which are to be found in the all too many methods for curing stammering. Because of the removal of the dread of speaking, there are always a few cures irrespective of the method used:

1. Economize the breath during speech. (2000 years old.)
2. Inhale before every difficult word. (1000 years old.)
3. Practice reading in a strong loud whisper.
4. Strengthen the laryngeal muscles by shouting in a loud voice and by the use of inflection exercises.

[1] The secondary manifestations are so numerous that Ssikorski (1891) devoted 143 pages of his book to an attempt to correlate them. Bluemel quotes the following from Quain's *Dictionary of Medicine*, 1st ed., p. 1513, "The paroxysms of stammering are the result of the subject's misdirected efforts to overcome his difficulty. In a typical case the mouth opens widely and remains in that position, the muscles of expiration and of the face and lips work convulsively, and the face becomes congested and the veins dilated and only after the patient ceases in his attempt to talk does the paroxysm come to an end." Some additional secondary or accessory movements are:

Movements of the arm, lifting the shoulders
Quick turning of the head to the side, and
Sometimes only a fixed sidewise glance, etc.

5. Control voluntarily the vocal organs.
6. Lift weights placed on the abdomen.
7. Contract the abdominal muscles and relax the diaphragm.
8. Inhale only through wide open mouth.
9. Practice special articulatory exercises.
10. Practice talking while walking uphill.
11. Speak always in a monotone.
12. Breathe always through the nose.
13. Practice exercises involving change of pitch.
14. Speak only in a whisper.
15. Practice holding the breath.
16. Drawl your words.
17. Speak in a low-pitched voice.
18. Count 1 to 50 with each breath.
19. Relax or yawn before speaking.
20. Begin talking in a very quiet voice.
21. Prolong only the initial vowels.
22. Accentuate differently placed words.
23. Intensify the vowels instead of prolonging them.
24. Read dialogues and relate anecdotes.
25. Strengthen the respiratory muscles.
26. Practice consonants and vowels before a mirror.
27. Never use a mirror in any kind of speech training.
28. Practice syllable and word exercises of every possible description.
29. Speak slowly for this prolongs the vowels.
30. Grunt before speaking.
31. Whistle before difficult words.
32. Gesticulate to withdraw attention. (100 years old.)
33. Converse with strangers—"stranger practice."
34. Use synonyms and circumlocutions.
35. Speak rhythmically with a metronome. (More than 100 years old.)
36. Control consciously the respiratory current.
37. Interpolate words and use "starters."
38. Emphasize each syllable.
39. Keep tip of tongue near the palate.

40. Imitate another person's voice.
41. Clench the fists at difficult words.
42. Refrain from speaking for long periods.
43. Do spontaneous talking.
44. Cultivate the memory—recite by heart.
45. Study physiology of speech elements.
46. Increase lung capacity.
47. Use dieting and drug therapy.
48. Practice vocal gymnastics with dumb-bells.
49. Take pedagogical training.
50. Follow Christian Science, Osteopaths and Chiropractors.

Mechanical Appliances.—Electrical apparatus is attached to a belt worn next to the body. When inclined to stammer, press button and get a shock. The galvanic, faradic and static electrical treatments have been almost discarded but may at any time find extensive use, such as recently galvanizing nose and throat.

Bate's silver tube for the mouth.
Itard's tongue fork. (1817.)
Colombat's "refoule langue" (tongue presser).
Wutzer's "glosso-mochlion."
Mouth full of gravel, after Demosthenes.

Surgical Methods.—In 1841, the operating craze began. Nearly 200 stammerers in France and 60 in Berlin were operated on in one year. "Cutting a transverse wedge from the base of the tongue. The edges of the gap sewed together, giving the tongue a high position. Also related operations." Medical means have also been applied with curative pedagogies: Belladonna, opium, potassium iodide, hydropathic remedies, peppermint oil and chloroform, croton oil, etc.

Psychological Methods:

Auto-suggestion.
Psycho-analysis.
Hypnotism.
Practice of internal speech.
Period of silence before treatment, etc.
Psychopedagogical methods.

From the foregoing list of instructions, it is readily seen that

they are largely concerned with breathing. Ssikorski (1891) and many others since then have found that emotional shock is present in the majority of cases. All authorities agree that *fear* dominates this condition, and affects the breathing. Normal quiet breathing has a calming effect on the emotions. The contrast is observed in fear, where breathing is inhibited and irregular, inspiration is frequently broken, a series of short breaths is followed by one or more deep ones, inspiration is short and expiration is prolonged. It is also apparent that fear aggravates physical stammering through disturbances in respiration. This disturbance is usually seen as an incongruity between the abdominal and chest breathing. Bluemel stresses the fluttering of the diaphragm. To confirm this, I observed by means of a fluoroscope the action of the diaphragm during the act of stammering.[1] The diaphragm appeared to me to quiver while remaining in a fixed position. Bluemel (1913) considered that the irregular action of the diaphragm is due to emotion rather than to physical stammering as shown by the fact that it is present before the stammerer begins to speak. Often it commences as soon as the stammerer is addressed.

These respiratory disturbances that accompany stammering have been investigated by many. Gutzmann and Liebmann (1895) used Marey's pneumograph and found that the respiration of most cases becomes irregular just before speech begins and that all kinds of abnormal inspiratory and expiratory movements occur during speech itself.

But it seemed to me that in a condition where the observable symptomatology is as diverse and infinite in variety as it is in stammering, that a spirographic study of the stammerer during mental repose—when relaxed—would show a reduced number of symptoms and thereby might perhaps indicate a fundamental departure from the normal. Therefore it became necessary to consider the subject of normal respiration, involving the factors of vital capacity, the so-called "dead air space" and tidal ventilation.

[1] Dr. John T. Farrell, Jr., roentgenologist at the Jefferson Hospital operated the fluoroscope and suggested making long exposure *x*-ray photographs of the diaphragm during the act of stammering.

RESPIRATION.

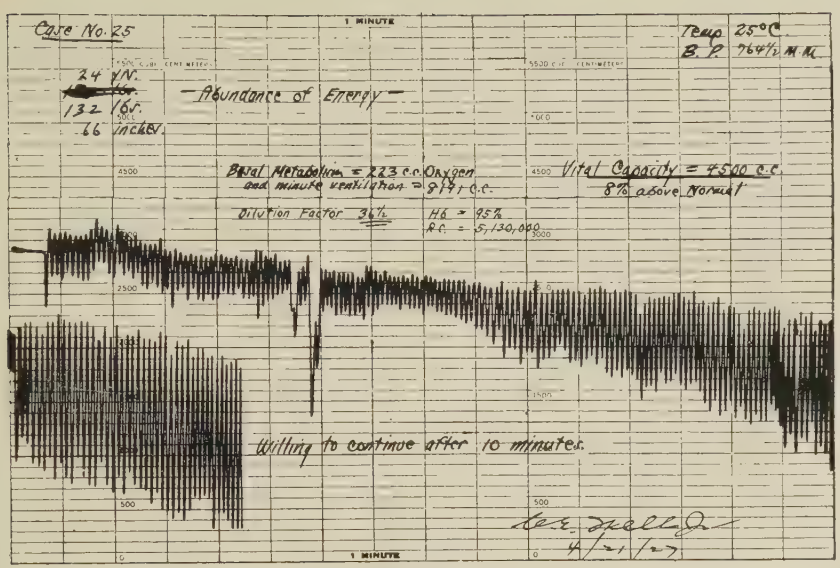
Normally, we give our breathing no thought. No doubt the inexhaustible abundance of air and the automatic character of respiration account for this. But when we consider, as Professor Brubaker has stated, that "we are standing at the bottom of an ocean of air 7 miles high at a pressure of 15 pounds to the square inch," and that the body cannot store enough oxygen to keep it alive even for ten minutes, then we begin to realize that breathing is a subject deserving of much thought.

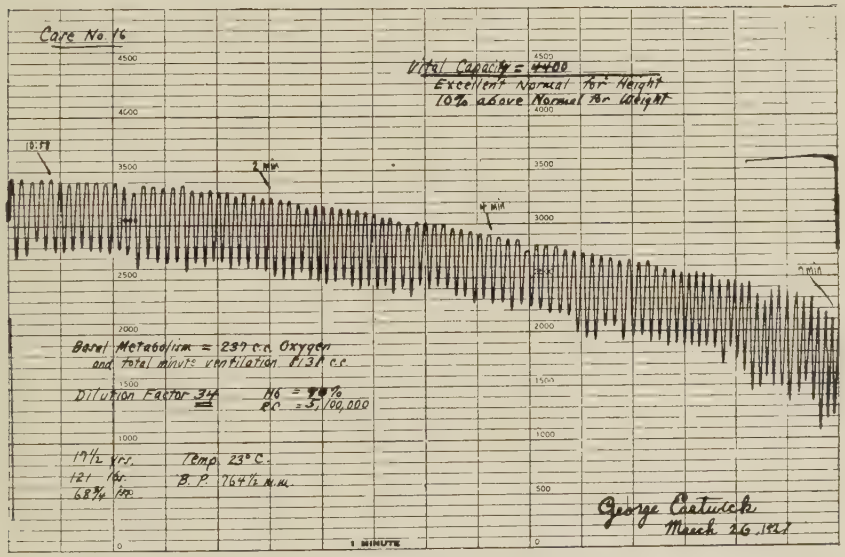
It is now recognized that breathing cannot be estimated merely by inspection.[1] "A doubling of the volume breathed per minute is scarcely or not at all noticeable either by the breather himself or by the casual observer, and it may even escape the careful observer, unless he measure it. Many of the impressions which we derive from our own feelings and are therefore inclined to accept as obvious truths, are in reality misleading or even directly contrary to the actual occurrences. For example: One who feels that the wind has been knocked out of him by a blow over the stomach and that he cannot inspire, although he strives to do so, is in fact inspiring excessively and is actually incapable for the moment of making an adequate expiratory movement. His lungs are not empty, but full."[2] This same effect was noticed in a few stammerers when stimulated with increasing amounts of carbon dioxide. Case No. 28, at the bottom of page 19 shows the sharp decline in the breathing curve in five minutes, from 3000 cc. to 1500 cc. This indicates that the air in the lungs was increased by 1500 cc. which was responsible for the subject's complaint that he was choking when in reality his lungs were not empty but full. (The normal response to increasing amounts of carbon dioxide is illustrated at the top of page 19.)

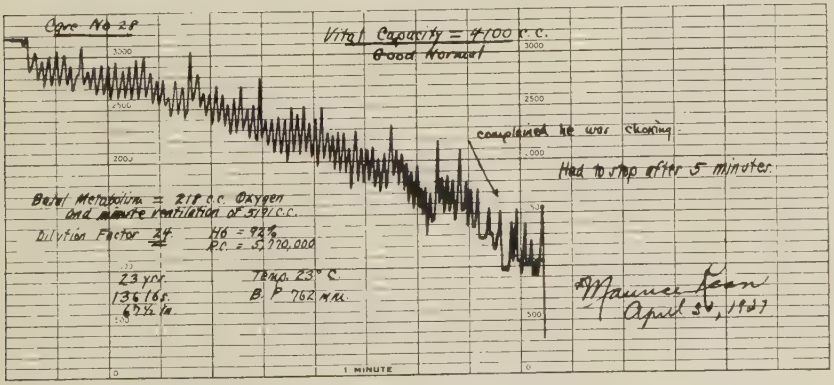
It is therefore evident that some measuring device is essential for obtaining quantitative data. I chose the Sanborn apparatus

[1] Edsall (1912) made clinical studies of respiration. Means and Mudd (1925) demonstrated the value of quantitative data in respiratory studies.

[2] Henderson and Haggard (1927): Noxious Gases and Respiration, etc.


Case No. 25
1 MINUTE
Temp 25°C
B. P 764½ M.M.
24 y/N.
132 lbs.
.66 inches.
Abundance of Energy
Basal Metabolism = 223 c.c. Oxygen
and minute ventilation = 9197 c.c.
Vital Capacity = 4500 c.c.
8% above Normal
Dilution Factor 36%
H6 = 95%
R.C. = 5,130,000
Willing to continue after 10 minutes.
Lee Spalding
4/21/27


Case No 16
Vital Capacity = 4400
Excellent Normal for Height
10% above Normal for Weight
10 FT
2 MIN
4 MIN
3 MIN
Basal Metabolism = 237 c.c. Oxygen
and total minute ventilation 8131 c.c.
Dilution Factor 34
H6 = 98%
R.C. = 5,100,000
19½ yrs.
121 lbs.
68¾ ins.
Temp 23° C.
B. P 764½ M.M.
George Cretucke
March 26 1927
1 MINUTE


Case No 28
Vital Capacity = 4100 c.c.
Good Normal
complained he was choking
Had to stop after 5 minutes.
Basal Metabolism = 218 c.c. Oxygen
and minute ventilation of 5179 c.c.
Dilution Factor 34
H6 = 92%
R.C. = 5,770,000
23 yrs.
135 lbs.
62½ ins.
Temp 23° C
B. P 762 MM.
Maurice Kean
April 30, 1927
1 MINUTE

because, though primarily designed for determining the basal metabolic rate, the graphs obtained also lend themselves readily to the calculation of volume per breath, respiratory rate and total ventilation per minute.

In using this metabolic apparatus, I followed all the standard basal conditions in every detail with a single exception, namely, that the subject was placed in a canvas semi-reclining chair at an angle of about 45 degrees. This position was not only restful, but made it possible for the subject, whenever unable to relax completely, to look at some non-exciting reading matter. This directed the attention of the patient away from the test. In every case, a trial test was made at first to acquaint the subject with the procedure and to insure a state of mental calm equally essential to an accurate metabolic determination and to spirographic records. In the hyper-excitable stammerers, two graphs of eight to ten minutes each were obtained, rather than one continuous test of fifteen to twenty minutes.

I found this semi-reclining position to be most satisfactory. Haldane and Douglas (1918) observed in their classical studies on shallow breathing that the effect of lying down is to accentuate the incomplete expansion of the lungs. I made a number of tests in the two positions and found one group that showed practically no difference, while for another group, lying down interfered with the expansion of the lungs.

Starr (1922) pointed out that Twitmyer had noted for many years that a large number of stammerers have very little chest expansion and were termed "sub-breathers." Consequently I deemed it necessary to secure spirographic records under standard basal metabolic conditions, and I therefore made all respiratory studies in the morning before breakfast after the subject had rested in the canvas reclining chair from forty to sixty minutes. Having a uniform method afforded an accuracy of comparison of cases that had not been possible heretofore. For example, though Gutzmann and Liebmann had made pneumographic tracings of stammerers in Vienna in 1895, Green in this country in 1901, and many others since then, all had overlooked the fact that there are conditions such as compensatory emphysema,

asthma, etc., in which the chest expansion is excellent but the vital capacity is greatly reduced. Pneumographic tracings show the excursions of the thoracic and abdominal walls, but do not indicate the volume of air which enters and leaves the lungs. Today we know that chest mobility is not an accurate index of the volume of the respiration. Lane (1921) showed that when an individual passes from extreme inspiration to extreme expiration, the alteration in the curve of the spinal column is a more marked feature than the elevation and depression of the costal arches. Dally (1923) found that increased thoracic mobility is not necessarily accompanied by increased chest measurement. The various workers who made pneumographic tracings further failed to take the precaution of requiring the subject not to take any food for at least several (preferably twelve to fourteen) hours previous to obtaining their tracings. This is essential, for Moersch (1925), working at the Mayo Clinic, found that among other factors, the vital capacity is influenced by the time elapsed since the last meal. Before taking up some of the many details of respiration, it will be necessary first to become familiar with the subject of vital capacity and also the question of the so-called "Dead Space."

Vital Capacity.—The "vital capacity" is the greatest amount of air that can be expired after a maximum inspiration. Myers (1925) states that it is a test of physical fitness insofar as the heart and lungs are involved. In my study, the vital capacity[1] was taken (the highest of three trials), with the subject standing and the spirometer[2] placed high to prevent stooping. Peabody and Wentworth (1917) found that there was a difference of 12 per cent by volume in 2 cases when standing and when "slumping," thus showing the importance of the standing position in obtaining the vital capacity. I used the standard values according to height and weight given in Myers' book on "Vital Capacity" (1927). Dreyer (1921) also has excellent tables classified into three groups according to occupation, but these tables were not

[1] Care was always taken to explain the necessity of taking as deep a breath as possible.

[2] Collins' spirometer was used.

practicable for my cases, 90 per cent of whom were students varying greatly from one another in their physical activities. The vital capacity varies with height, weight, sex, age, and skin area. The advantage of a large vital capacity is that the air in the lungs need be replaced less frequently. Also, this usually means a larger respiratory surface with the less likelihood of shortness of breath during active exercise, for the exchange of gases takes place over a larger surface. Thus the bellows or lungs have less work to do. The vital capacity does not represent the actual capacity of the lungs for there is always a certain amount of air remaining in the lungs after the most forceful expiration. This air is known as the residual air. This, plus the vital capacity, equals the total capacity of the lungs. In normal breathing, only 10 per cent to 12 per cent of the vital capacity is used. This is known as the "tidal ventilation." Vital capacity represents the functional capacity while the total capacity of the lungs represents merely anatomic capacity.

Dead Space.—Before an attempt is made to interpret the effects of the Vital Capacity on breathing, it will be necessary to understand the influence of the so-called "dead space." The anatomic[1] dead space was found by Loewy to be 144 cc. in a cadaver. This space includes the naso-pharynx, oropharynx, trachea, large and small bronchi and has been approximated from 100 to 175 cc. by several investigators. Theoretically, this amount of air, or at least a large portion of it, should be deducted from each respiration for the air entering the dead space on inspiration promptly passes out again on expiration, with very little gas exchange. Thus, it is apparent that the size or volume of the dead space is important, particularly so in the rapid and shallow type of breathing. Unfortunately, there is no agreement among authorities as to whether the dead space is fixed in size for a given individual or varies with deep, moderate and shallow breathing. Edsall (1913) states that this space is a waste as far as any direct influence upon gas exchange is concerned, and more so in rapid, shallow breathing. He illustrates this by considering

[1] The anatomic and physiologic dead space are not identical for as Yandell Henderson of Yale has demonstrated—*Gases passing through tubes do so by flowing through the center.*

the individual who breathes very slowly but deeply four times in a minute, taking 1800 cc. at each inspiration, which gives a total minute ventilation of 7200 cc. Deducting 140 cc. (dead space) for each of the four respirations or 560 cc. from the total of 7200 leaves 6640 cc. of real or alveolar ventilation. In contrast, take the rapid, shallow breather, having an equivalent total ventilation, but with a respiratory rate of 32, taking only 225 cc. at each inspiration. The total ventilation equals 7200 cc., but 4480 cc. must be deducted (140 cc. $\times$ 32) from the total ventilation, leaving only 2720 cc. as the real or alveolar ventilation. Thus, slow, deep breathing gives 144 per cent more alveolar ventilation. Krogh believes that the depth of respiration has but little effect upon the size of the physiologic dead space. Pearce and Hoover (1920) found that at moderate volumes of inspiration, the dead air space was a relatively fixed volume. Macklin (1925) in his careful *x*-ray studies on bronchial movements notes that there is an elongation of the trunk and all the branches of the bronchial tree in inspiration and a corresponding shortening of these structures in expiration. Mudd (1925) contends that small respirations under 100 cc. do not affect the alveolar ventilation for this volume is less than the dead space calculated by several observers. Means (1924) points out that it is both the ventilation of the dead space and the alveoli that determines the magnitude of the total ventilation, thus indicating the importance that the size of the dead space plays in the mechanical efficiency of breathing. Heinbecker (1927) states that within the limits of quiet respiratory excursions there is practically no change in the caliber of the bronchi except during the early part of inspiration when some of the larger bronchi, particularly those leading to the lower lobes, are actually narrowed.

Henderson, as well as Haldane, is of the opinion that the size of the dead space increases with deep respiration and decreases with shallow or rapid superficial respiration. These differences of opinion arise from the fact that the size of the dead space can be determined only by indirect methods. For instance, Haggard and Henderson (1927) determined the dead space by analysis of mixed expired air. Ethyl ether gas was inhaled in low con-

centration and since it is highly soluble in blood, nearly all of it that reaches the lungs is absorbed. The expired air contains approximately 33 per cent of the original amount of ethyl ether, which comes from the dead space. Thus they conclude "that the finer air tubules in the lungs and perhaps even the centers of the air sacs themselves may contribute to the dead space. These air sacs are elastic and are enlarged by deep breathing; the functional or so-called 'virtual dead space' is not a constant absolute volume, but is relatively about one-third of the volume of the breath both in deep and moderate breathing." I have thus reviewed the subjects of vital capacity and dead space in order to show their relation to that function with which the stammerer is concerned—his respiration.

Respiration is under dual control. The movements of respiration, although they may be under voluntary control for short periods, are in the main involuntary and automatic. The principle stimulus depends on the chemical changes in the arterial blood. Thus it may be observed that one can temporarily inhibit the respiratory center by holding the breath, but the carbon dioxide accumulates and this stimulus becomes so great that taking a breath cannot any longer be inhibited. In this way, breathing is removed from voluntary control. However, the type of respiratory movements is maintained and coördinated by afferent impulses constantly passing up the vagus nerves from the lungs themselves. These afferent impulses are initiated in inspiration, when a definite tension in the air vesicles of the lungs is reached, but the moment at which the actual conversion of the inspiratory phase into an expiratory phase becomes effective, is dependent upon the extent of the chemical stimulus and the sensitivity of the respiratory center. Any increase in the amount of carbon dioxide is accompanied by an increase in the depth of respiration, while a deficiency of oxygen or anoxemia causes an increase in the respiratory rate with shallow breathing. In the latter instance, respiration is not efficient, as has already been considered in detail under the heading of "dead space." Thus the chemical control of breathing determines the amount of ventilation, but the vagus controls the type of breathing that is to accomplish the necessary ventilation.

Ordinarily the magnitude of the total ventilation depends on the metabolic demand. Henderson and Haggard have formulated the "first law of breathing," namely, that pulmonary ventilation will be proportional to the metabolic rate. But we shall see many exceptions to this rule, in the case of these stammerers. The work of Krogh and Lindhard (*Jour. Physiol.*, 1913) suggests that the nervous and mental factors also play a part in determining the total ventilation of the lungs. They found that when subjects were told to begin work and expected a heavy load, the same increase in ventilation and blood flow occurred even though there was actually no load on the work apparatus.

The average adult consumes from 400 to 600 liters of oxygen per day, depending primarily on the individual's activity, although the exact amount depends on weight, height, age, sex and the metabolic rate. Taking 500 liters of oxygen as an average and multiplying by the average normal dilution factor[1] of 25, gives a total of 12,500 liters of air breathed daily. We can thus see at a glance the significance of the dilution factor. For example, an increase in the dilution factor of 5 per cent would give a total of 15,000 liters. This will be considered in detail later when presenting the findings in stammerers.

In the average resting man, about 500 cc. of air is inspired with each breath, and almost the same amount is expired with each breath, during ordinary, quiet breathing. This is known as the "tidal ventilation." Haggard and Henderson's range is from 300 to 600 cc., the former figure no doubt is low so as to include the tidal ventilation for women. Burns' (1921) figures are from 440 to 550 cc. Schneider (1922) found the average depth of respiration to be 570 cc. in man. Mitchell (1923) states that when measured with a spirometer, the tidal air is around 500 cc. Brubaker (1927) states the normal range is from 330 to 500 cc. My own experience in measuring the volume per breath or tidal ventilation in about 100 normal cases approximates in the case of women the lower figure of Professor Brubaker, while with men it is around 500 cc.

[1] The dilution factor represents the exact amount to which oxygen is diluted by the air breathed.

The Significance of Shallow Breathing.—The normal rate and depth of respiration are dependent upon the maintenance of a normal balance between chemical and nervous stimuli. From the chemical standpoint, the chief causes of variation from the normal respiratory rhythm are alterations in the pressures of oxygen and carbon dioxide in the arterial blood or tissues. Such alterations, if they be primary factors in the causation of shallow breathing, must be dependent either upon an abnormal concentration of oxygen or carbon dioxide in the inspired air or upon the presence of some diseased process in the organism, most commonly respiratory, circulatory or metabolic in nature. The fact that such conditions are not, as a rule, operative in the group of patients under observation necessitates that we turn to a consideration of the nervous control of respiration for a possible explanation of the cause of the shallow breathing which was noted in these cases.

The nervous apparatus concerned with respiratory functions consists of two divisions, central and peripheral, the former being the respiratory center in the floor of the fourth ventricle, the latter consisting of fibers contained in the trunks of the vagus nerves. The normal respiratory rhythm is the resultant of the normally balanced activities of these two mechanisms, a disturbance of this balance being manifested by certain definite alterations in the rate and depth of respiration. Thus, stimulation of the respiratory center, as by increased CO_2 pressure or by increased hydrogen-ion concentration causes an increase in depth and decrease in rate of respiration. The same results follow section of both vagus nerves. These phenomena are an expression of central preponderance, due in the first case, to central stimulation, and in the second, to destruction of the peripheral mechanism, the end-result in both instances being a similar disturbance of balance.

On the other hand, direct stimulation of the vagus nerves, as by the electrical current, results in an increased rate and decreased depth of respiration. The same type of breathing follows fatigue of the respiratory center as demonstrated by Haldane. These phenomena are an expression of vagal or peripheral preponder-

ance, due in the first case, to direct stimulation and in the second to release from central control.

Thus, shallow breathing may be an indication of a disturbance of the balance between the two divisions of the nervous mechanism of respiration, resulting from either depression of the activity of the respiratory center or hyper-activity or hyper-irritability of the vagus fibers. It must also be remembered that the rate and depth of respiration are to a limited extent under voluntary control. However, the prolonged maintenance of an abnormal rhythm results in alterations in the pressures of oxygen and carbon dioxide in the arterial blood which removes the breathing from voluntary control and eventually the abnormal rhythm becomes for a given individual a fixed type of breathing.

HEMATOLOGY

Following the spirographic tests, hematologic studies were made on each case. Thus the hemoglobin estimations and blood counts were determined under standard basal metabolic conditions hereinafter described.

Hemoglobin.—Haden (1922) emphasized that all hemoglobinometers were standardized to read in per cent and yet there was no agreement as to the number of grams of hemoglobin which are represented by 100 per cent. We know that 100 per cent should be the amount of hemoglobin contained in 100 cc. of an average normal blood, assuming the red cell count to be 5 million. In order to determine the average hemoglobin content of blood for 100 cc. for each 5 million cells, Haden counted the erythrocytes and estimated with the Van Slyke method the hemoglobin of 52 normal persons. In accordance with his high standard of accuracy, he recalibrated his glassware. The hemocytometer and the blood counting pipets were checked by the Bureau of Standards. He found the average hemoglobin content of men (eighteen to fifty years of age) to be 15.6 gm. for 100 cc. of blood.

The fact that men, women and children have different amounts of hemoglobin ranging from 14 to 17 gm. was stressed by Professor Starr in his seminar on Metabolism and Behavior. Likewise the variations in the different hemoglobinometers were considered as well as diurnal variations.

Kern (1924) likewise stressed the fact that 100 per cent hemoglobin is equivalent to values from 13.77 to 17.3 gm. depending on the hemoglobinometer used. Brown and Rowntree (1925) admit there is no agreement as to the normal standard for hemoglobin. Since the Van Slyke method is now the generally accepted standard procedure to calibrate the different hemoglobinometers, the only question that remains is the number of grams of hemoglobin that shall represent 100 per cent.

In this work, after having calibrated the Sahli-Leitz hemoglobinometer on the basis of Haden's figure of 15.6 gm. and the Newcomer on the basis of Williamson's figure of 16.9 gm., I

averaged these and the figure 16.25 gm. was used in this investigation as equivalent to 100 per cent.

Since this essay is a hemato-respiratory study, it is appropriate to consider some of the factors which influence, especially those which increase, the number of erythrocytes in the blood stream per unit-volume. But it will not be necessary to discuss exhaustively what constitutes a normal red cell count. Because of the inherent variations in technique and of those caused by the personal equation, there cannot be an absolute normal figure. In addition, there are diurnal variations and possibly seasonal ones.

Herewith are submitted average normal erythrocyte counts according to:

Brubaker.		*Sahli.*	
Men	5.0 million	Men	5.0 million
Women	4.5 million	Women	4.5 million
Gram.		*Haden* (venous blood).	
Men	5.37 million	Men (18 to 30 years),	5.080 million
Women	4.78 million	Men (30 to 50 years),	4.865 million
Gram and Norgaard.		*Osgood* (venous blood).	
Men	4.85 million	Men (18 to 30 years)	5.39 million
Women	4.36 million	Women (18 to 30 years)	4.80 million

Many factors beside altitude may increase the number of red cells per unit of volume. An increase follows: active exercise, cold baths, partaking of food, massage, asphyxia and fright. Cannon has shown that asphyxia causes an output of epinephrin into the blood and this may be a contributory cause of the increased red cell count. Ferrari in 1897 found in students taking examinations, a slightly increased red cell count, possibly due to fright or anxiety. From my own observations of medical students, I consider it probable that they were similarly affected especially when several cubic centimeters of venous blood were withdrawn. A marked rise in erythrocytes has been noted following active exercise. This fact has recently been applied in improving the accuracy of blood volume determinations by having the patient exercise to insure that the injected dye is distributed through the circulation. Even psychologic processes

implying exercise were found by Barcroft and Stephens (1927) to be followed regularly by a decrease in the size of the spleen with an increase in the number of red corpuscles.

A variety of conditions, among which may be mentioned organic and congenital heart disease, emphysema, bronchial asthma, dyspnea and any condition impairing the circulation or aëration may compensate by an increase in hemoglobin or in red cells or in both. For instance, an obstruction of the vena cava will result in an increase in the number of erythrocytes. Any form of even temporary anoxemia will have the same result.

Persistent vomiting or profuse sweating or protracted diarrhea will dehydrate the blood. This condition was likewise found following war gas-poisoning (chlorine, chlorpicrin, mustard or phosgene).

Red Cell Counts.—These were obtained by pricking the finger deeply enough to allow the blood to flow freely. Duplicate counts not varying by more than 200,000 were made in each instance. In order to prevent being unconsciously influenced to obtain counts which check closely, a mechanical counter[1] was used. Such a counter records each cell without letting the operator know what the total will be until he has completed each count. He is therefore not influenced by the amount of any previous count. The Bureau of Standards certified pipets and Levy-Hausser counting chamber (B. S. No. 4304) were used.

Leak, Kohl and Stebbins (1927) found that the average maximum diurnal variation in the red count for men was 345,000 and for women 310,000. In order to reduce to a minimum the discrepancies caused by these diurnal variations, it was decided to make all blood counts at a fixed time of day, namely, between 10 and 11 A.M., before breakfast. In many cases which showed an erythrocyte count above 6 million, additional counts were made later in the afternoon. This time of day has been found by Dreyer (1919) to show the smallest daily variation in hemoglobin. In some cases the erythrocyte count was checked by a count made on venous blood. In a few cases the blood counts were obtained from the lobe of the ear, but this method was not

[1] Hand tally counter—Veeder Mfg. Co., Hartford, Conn.

found satisfactory as it required an undue amount of squeezing which caused inaccuracies in the count.

Finally, I made it a practice to obtain the blood count after the necessary rest required for a basal metabolic determination. This also helped to avoid the extremes of outdoor temperature.[1]

Red Cell Volume.—This was determined by the Van Allen hematocrit tube. This hematocrit is a graduated instrument by which the volumetric relationship between corpuscles and plasma is determined. A free flow of blood upon the skin surface of the finger-tip was obtained by a needle prick, and the first two drops discarded. A measured volume of blood was collected to the 100 mark of the graduated tube, then diluted with 1.3 per cent sodium oxalate, and sealed with a spring clip. This determination was made in duplicate. Then the two hematocrit tubes were centrifuged at a minimum rate of 2700 r.p.m. to a constant volume. Thus the red corpuscles became separated from the plasma by virtue of a difference in their specific gravities and the red cell volume reading was made directly from the graduated tube.

The normal range for the red cell volume in man is from 42 to 50 per cent, and in woman from 39 to 43 per cent.

White and differential counts were made in all cases. The only significant finding was an increase in eosinophiles in two of the cases with emphysema.

This then completes the consideration of the hematologic principles. Having already treated the general facts of stammering and the principles of respiration underlying this condition, it now remains to present the data obtained in this investigation.

[1] It is common knowledge that the limbs swell during warm and shrink during cold weather. Bazett (1928).

DATA ON THE 101 CASES OF STAMMERING.

I have numbered the cases consecutively as they came to the Speech Clinic, but have grouped the adult cases according to the red cell totals and the hemoglobin content.

Group A contains the cases whose red cells numbered from 5,900,000 to 7,360,000 per c.mm. with an average hemoglobin content of 95 per cent (100 per cent equivalent to 16.25 gm.).

Group B contains the cases whose red cells numbered less than 5,000,000 per c.mm. with an average hemoglobin content of 85.5 per cent.

Group C contains the cases whose red cells numbered from 5,000,000 to 5,900,000 per c.mm., with an average hemoglobin content of 90 per cent.

No attempt was made to apply this classification to the subjects under fifteen years of age, because of the lack of hematologic standards for children. However, the findings on the juvenile cases are presented as Group D, but no deductions made from them, since children vary too greatly in physical development to determine the norm for ventilation per minute, volume per breath, oxygen dilution factor.

After the above data had been obtained and the calculations made, I found my hypothesis verified, namely, that *the cases exhibited a correlation between the type of breathing and the red cell count and hemoglobin content—a correlation which may be explained as a principle of physiologic compensation.* The stammerer whose breathing was shallow compensated by a high red count and high hemoglobin. In other cases, periodic deep breaths were taken in a more or less successful attempt to assist in the compensation. Likewise, many cases showing low vital capacity compensated by an increase in the depth of breathing. When the compensation was adequate, the red cell count and hemoglobin were normal, where incomplete the erythrocytes and hemoglobin were increased. Finally, in other cases, the breathing was deep with normal blood findings.

GROUP A. —17 ADULT CASES WITH RED CELLS 5,900,000 TO 7,360,000.

Case No.	Age.	Wt.	V. C., per cent.	Vol. per breath.	R. R.	Tot. vent. in liters.	O. per min. in cc.	Oxygen dil. fac.	Hb. %.	Red cells.	R. C. vol.	B. M. R.
45 . .	18	164	N	376	16	6.122	274	22	98	6,370,000	52½	− 1
50 . .	19	130	N	502	15	7.646	223	34	95	6,200,000	54	− 5
85 . .	19	182	N	784	12	9.045	314	29	100	6,410,000	51	+11
94 . .	17½	120	N	371	16	6.045	254	24	100	6,520,000	48	+10
97 . .	17½	130	N	414	16	6.508	263	25	90	6,000,000	46	+ 4
9 . .	17	130	N	274	22	5.941	239	21	90	6,000,000	50	− 1
28 . .	22	136	N	377	14	5.191	218	24	92	5,940,000	45	− 7
100 . .	15	135	− 5	429	14	5.932	286	21	95	5,930,000	48	+ 1
75 . .	24	155	−11	459	13	6.066	240	25	102	6,630,000	51	N
11 . .	17	162	+15	423	16	6.556	275	24	97	5,900,000	49	+ 1
14 . .	23	133	+10	397	14	5.437	250	22	89	7,360,000	49½	+ 8
60 . .	19½	173	+ 5	404	14	5.734	257	24	85	6,150,000	....	− 1
82 . .	19	123	+10	410	14	5.954	240	25	95	5,950,000	45	+ 4
12 . .	20	100	−20	320	16	5.262	200	26	95	6,070,000	50	N
80 . .	17	132	−20	367	17	6.355	258	25	98	6,390,000	48	+ 5
53 . .	18	156	−22	548	13	7.018	240	29	97	6.040,000	47	− 2
5 . .	19	150	....	384	15	5.754	297	19	95	6,000,000	....	+19
Average .	19	142	− 2	426	15	6.268	254	24	95	6,224,000	49	+ 3

V. C. = Vital capacity (N = Normal).
R. R. = Respiratory rate.
Tot. vent. = Total ventilation.
O. per min. = Oxygen consumed per minute.
Dil. fac. = Dilution factor.
R. C. vol. = Red cell volume.
Hb. 100% = 16.25 gm. Hb. in 100 cc.
B. M. R. = Basal metabolic rate.
Total ventilation is the same as total minute ventilation.

GROUP B. —19 ADULT CASES WITH RED CELLS BELOW 5,000,000.

Case No.	Age.	Wt.	V. C., per cent.	Vol. per breath.	R. R.	Tot. vent. in liters.	O. per min. in cc.	Oxygen dil. fac.	Hb. %.	Red cells.	R. C. vol.	B. M. R.
44 ♀	23	114	+25	591	12	7.264	205	35	87	4,680,000	38	+ 5
1	21	173	+15	549	11	6.144	271	23	85	4,710,000	..	− 2
37 ♀	15	100	+10	526	16	8.200	250	33	82	4,780,000	38	....
7	19	158	+ 5	726	10	7.126	220	33	85	4,790,000	..	−14
48	17	130	N	374	17	6.412	263	24	88	4,830,000	..	+ 4
49	22	160	N	678	12	8.120	202	40	86	4,860,000	45	−21
86	18	140	N	511	18	9.191	250	37	91	4,750,000	45	N
4	15	76	N	374	23	8.410	172	49	83	4,400,000	37	+10
40	17½	125	N	628	9	5.804	220	26	88	4,730,000	45	− 8
41	21	135	N	380	19	7.299	208	35	80	4,660,000	42	− 7
39 ♀	20	95	−17	305	14	4.261	181	23	75	4,250,000	37	+ 1
90	23	137	−38	584	11	6.591	211	31	88	4,370,000	42	− 9
58 ♀	17½	108	− 7	415	12	4.884	187	22	85	4,100,000	44	− 2
84 ♀	15	99	− 7	336	16	5.220	183	29	81	4,980,000	43	−13
42	24	107	− 6	454	17	7.604	210	36	87	4,700,000	43	+11
93	17	120	−10	344	16	5.502	242	23	85	4,710,000	40	+ 2
67 ♀	16	115	−10	433	11	4.725	187	25	85	4,900,000	41	−11
35	17½	145	−15	393	18	7.052	280	25	90	4,300,000	41	+ 3
101	20	141	+12	609	11	6.840	179	38	94	4,850,000	48	−21
Average	20	125	− 2	485	14⅓	6.665	217	31	85½	4,650,000	41.7	− 4
Average of adult male cases	19	135	− 3	508	15	7.084	225	32	87	4,667,000	42.8	− 4

V. C. = Vital capacity (N = Normal).
R. R. = Respiratory rate.
Tot. vent. = Total ventilation.
O. per min. = Oxygen consumed per minute.
Dil. fac. = Dilution factor.
R. C. vol. = Red cell volume.
Hb. 100% = 16.25 gm. Hb. in 100 cc.
B. M. R. = Basal metabolic rate.
Total ventilation is the same as total minute ventilation.

GROUP C.—46 ADULT CASES WITH RED CELLS 5,000,000 TO 5,900,000.

Case No.	Age.	Wt.	V. C., per cent.	Vol. per breath.	R. R.	Tot. vent. in liters.	O. per min. in cc.	Oxygen dil. fac.	Hb. %.	Red cells.	R. C. vol.	B. M. R.
63	15	124	− 3	503	12	6.085	270	23	91	5,140,000	46	+ 4
33	18½	142	− 4	322	22	7.264	241	30	87	5,600,000	42	− 2
10	25	135	− 5	392	18	7.252	234	31	84	5,060,000	52	N
61	19½	115	− 6	364	14	5.106	196	26	95	5,540,000	47	− 7
2	19½	123	− 5	410	11	4.464	182	24½	83	5,150,000	...	− 7
47	20	130	− 8	417	15	6.259	217	29	97	5,690,000	52	− 7
27	16	120	− 9	322	16	5.061	221	23	96	5,558,000	44½	− 9
88	22	145	− 9	656	9	6.060	236	26	88	5,540,000	...	− 2
6	18	108	−10	347	16	5.441	187	29	75	5,020,000	...	N
15	17	125	−10	536	17	9.309	233	40	90	5,560,000	44	− 2
26	16	126	−10	394	14	7.988	237	34	85	5,370,000	44	− 9
32	26	158	−10	312	27	8.450	291	29	90	5,170,000	44	+12
72	17	130	−11	495	18	8.788	271	32	92	5,770,000	45	+ 6
79	21½	137	−11	526	10	5.386	255	21	96	5,600,000	47	+ 7
92	16	116	−12	441	16	7.082	237	30	90	5,830,000	46	− 3
57	32	125	−13	441	19	8.444	203	42	76	5,550,000	44	− 4
8	16	124	−14	344	19	6.558	235	28	93	5,400,000	48	− 4
20	17	132	−15	223	28	6.186	255	24	96	5,790,000	45	+ 3
23	20	135	−16	430	13	5.678	228	25	84	5,140,000	42	− 3
59	20	140	−16	423	18	7.464	224	33	93	5,560,000	...	− 5
99	17	140	−16	392	16	6.172	230	26	98	5,380,000	48	− 8
91	16½	102	−17	449	18	8.212	190	43	75	5,640,000	40	− 6
70	15	109	−18	291	21	5.910	230	26	90	5,630,000	42	− 4
38	26	172	−20	480	16	7.715	278	28	98	5,430,000	42½	+ 6

V. C. = Vital capacity (N = Normal).
R. R. = Respiratory rate.
Tot. vent. = Total ventilation.
Total Ventilation is the same as total minute ventilation.
P. per min. = Oxygen consumed per minute.
Dil. fac. = Dilution factor.
R. C. vol. = Red cell volume.
Hb. 100% = 16.25 gm. Hb. in 100 cc.
B. M. R. = Basal metabolic rate.

GROUP C. —46 ADULT CASES WITH RED CELLS 5,000,000 TO 5,900,000—(*Continued*)

Case No.	Age.	Wt.	V. C., per cent.	Vol. per breath.	R. R.	Tot. vent. in liters.	O. per min. in cc.	Oxygen dil. fac.	Hb. %.	Red cells.	R. C. vol.	B. M. R.
89 . . .	30	145	−22	678	8	5.755	216	26	95	5,740,000	45	−13
21 . . .	15½	126	−23	371	18	6.771	257	26	95	5,140,000	41	+ 1
16 . . .	17½	121	N	520	16	8.138	237	34	90	5,100,000	43	N
18 . . .	21	145	N	706	8½	5.918	257	23	76	5,850,000	40	+ 3
19 . . .	19	126	N	526	16	8.708	247	35	93	5,010,000	45	+ 8
24 . . .	20½	127	N	388	15	5.772	200	29	94	5,200,000	44	− 8
29 . . .	17½	125	N	387	15	5.871	247	24	95	5,520,000	47	N
31 . . .	20	160	N	502	12	5.714	250	23	84	5,760,000	46½	− 7
34 . . .	26	142	N	411	14	5.919	220	27	85	5,390,000	42½	− 6
43 . . .	22	155	N	832	11	9.326	244	38	89	5,430,000	43	− 1
52 . . .	15	153	N	485	16	7.979	272	29	85	5,140,000	49	−10
62 . . .	19	137	N	430	14	6.186	280	22	90	5,600,000	47	+ 5
65 . . .	23	164	N	461	16	7.200	270	27	90	5,270,000	43	+ 3
74 . . .	20	150	N	408	19	7.750	257	30	94	5,750,000	44	+ 2
81 . . .	16	111	N	428	14	5.715	210	27	93	5,610,000	47	− 8
17 . . .	20	141	+ 8	398	15	6.080	246	24	95	5,160,000	41	− 2
25 . . .	24	132	+ 8	419	20	8.191	223	36	95	5,130,000	44	− 2
30 . . .	16	148	+12	554	11	5.881	250	24	95	5,670,000	44	−10
56 . . .	22	182	+11	643	12	7.720	270	29	88	5,820,000	47	− 4
76 . . .	18	162	+16	636	8	5.265	280	18	94	5,530,000	45	+ 6
87 . . .	21	146	+10	435	15	6.532	209	31	90	5,820,000	48	−15
95 . . .	21	135	+ 5	388	15	5.818	269	22	90	5,690,000	42	....
Average .	20½	136	− 5	455	15½	6.750	238	28⅓	90	5,466,300	44.8	− 1

V. C. = Vital capacity (N = Normal).
R. R. = Respiratory rate.
Tot. vent. = Total ventilation.
Total ventilation is the same as total minute ventilation.
O. per min. = Oxygen consumed per minute.
Dil. fac. = Dilution factor.
R. C. vol. = Red cell volume.
Hb. 100% = 16.25 gm. Hb. in 100 cc.
B. M. R. = Basal metabolic rate.

GROUP D. —19 CASES OF BOYS AND GIRLS UNDER FIFTEEN YEARS.

Case No.	Age.	Wt.	V. C., per cent.	Vol. per breath.	R. R.	Tot. vent. in liters.	O. per min. in cc.	Oxygen dil. fac.	Hb. %.	Red cells.	R. C. vol.	B. M. R.
3 . . .	13	76	+10	404	10	8.322	190	44	76	4,350,000	40	+ 7
98 . . .	14½	100	+ 5	288	21	6.132	217	28	80	4,720,000	37	− 5
54 . . .	11	80	N	302	15	4.536	157	29	71	4,560,000	36	− 7
96 . . .	9	88	N	200	..	.....	...	...	87	4,700,000		
51 . . .	11	70	− 7	308	17	5.281	180	29	90	4,510,000	41	− 2
46 ♀ . .	14½	108	−15	287	15	4.361	178	24	80	4,240,000	40	− 1
13 . . .	12½	84	+10	457	16	7.431	210	35	90	5,270,000	48	+ 2
73 ♀ . .	13⅔	104	+ 4	389	21	8.065	218	37	85	5,470,000	42	+ 2
83 . . .	14	97	+12	328	19	6.195	201	31	83	5,520,000	40	− 9
55 ♀ . .	13	100	N	375	17	6.280	185	34	81	5,180,000	41	− 1
69 . . .	12	90	N	311	18	5.745	223	26	83	5,530,000	38	+ 6
71 . . .	14	113	N	340	15	5.049	200	25	81	5,650,000	39	− 5
64 . . .	13	93	− 5	268	22	5.879	206	29	83	5,250,000	41	− 4
22 . . .	14	92	−10	275	18	4.958	205	24	95	5,210,000	45	+ 4
68 . . .	13½	87	−20	307	16	4.910	191	26	78	5,200,000	40	+ 1
36 . . .	13	86	−35	304	18	5.472	219	25	84	5,450,000	38	+10
66 . . .	12	84	+25	355	14	5.061	198	25½	87	6,280,000	42	N
77 . . .	12½	119	− 7	296	20	5.755	265	22	87	5,920,000	42	+ 8
78 . . .	13½	100	−10	362	14	5.245	227	33	96	5,930,000	48	N

V. C. = Vital capacity (N = Normal).
R. R. = Respiratory rate.
Tot. vent. = Total ventilation.
Total ventilation is the same as total minute ventilation.
O. per min. = Oxygen consumed per minute.
Dil. fac. = Dilution factor.
R. C. vol. = Red cell volume.
Hb. 100% = 16.25 gm. Hb. in 100 cc.
B. M. R. = Basal metabolic rate.

The principle of hemato-respiratory compensation becomes evident through an examination of the *group averages.*

	Group A.	*Group B.*	*Group C.*
Number of adult cases	17	19	46
Age	19	20	20½
Weight	142	125	136
Respiratory rate	15	14	15½
Total ventilation per minute, in liters	6.268	6.665	6.750
Volume per breath in cubic centimeters	426	485	455
Oxygen dilution factor	24	31	28
Hemoglobin (per cent)	95	85½	90
Red cell volume (per cent)	49	41.7	44.8
Red cell count	6,224,000	4,650,000	5,466,000

In Group B there are 6 female cases, while there are none in Group A. Therefore, to make an accurate comparison it is necessary to eliminate the 6 female cases. Thus, the volume per breath for males in Group B averages 508 cc. instead of 485 cc., and the weight is 135 instead of 125. The total ventilation equals 7.084, the dilution factor 32 and red cells 4,667,000.

The age and respiratory rate of all three groups are so much alike that no comparison need be made in this respect. But in Group B, it can be seen that the total ventilation per minute, volume per breath, and the dilution factor are all larger than in Group A, even though the latter show an average weight of 17 pounds more than the average for Group B. One would expect the heavier individuals,[1] providing they were of normal weight, to have a larger volume per breath as well as a greater total ventilation per minute, because they need more oxygen than those of lighter weight. But such is not the case. In fact, Group B has a volume per breath 14 per cent above, a total ventilation per minute 6 per cent above, and a dilution factor 29 per cent above that of Group A. If the female cases are not counted, then the contrast is more striking, namely, volume per breath 19 per cent above; total ventilation 13 per cent above, and dilution factor 33 per cent above that of Group A. Thus, Group B demonstrates more efficient breathing than Group A, and, according to my theory, should not require any compensa-

[1] Only 4 cases in this investigation were overweight.

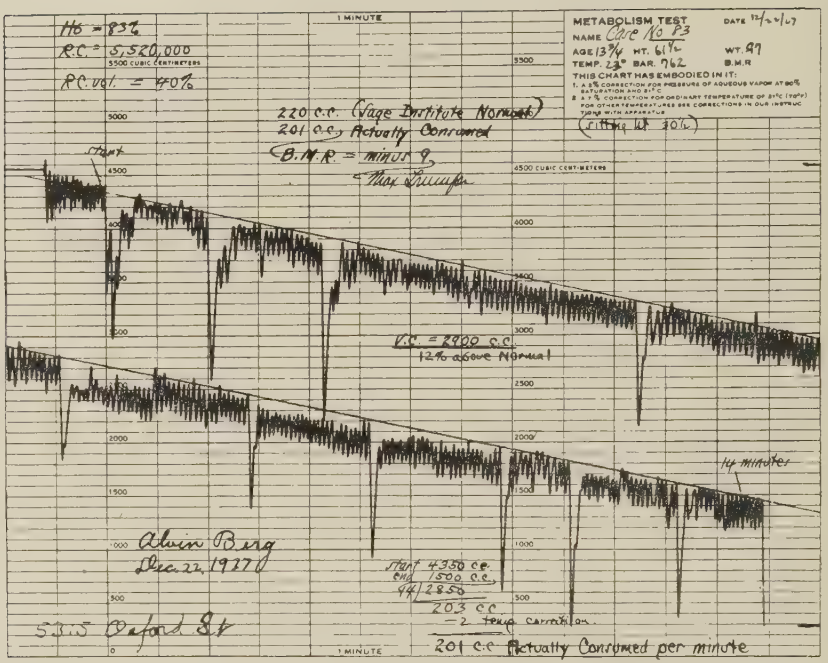
tory rise in red cells or hemoglobin. Such is the case. On the other hand, in Group A, the hemoglobin and red cells are high, because they are needed to compensate for the low volume per breath, low total ventilation and dilution factor.

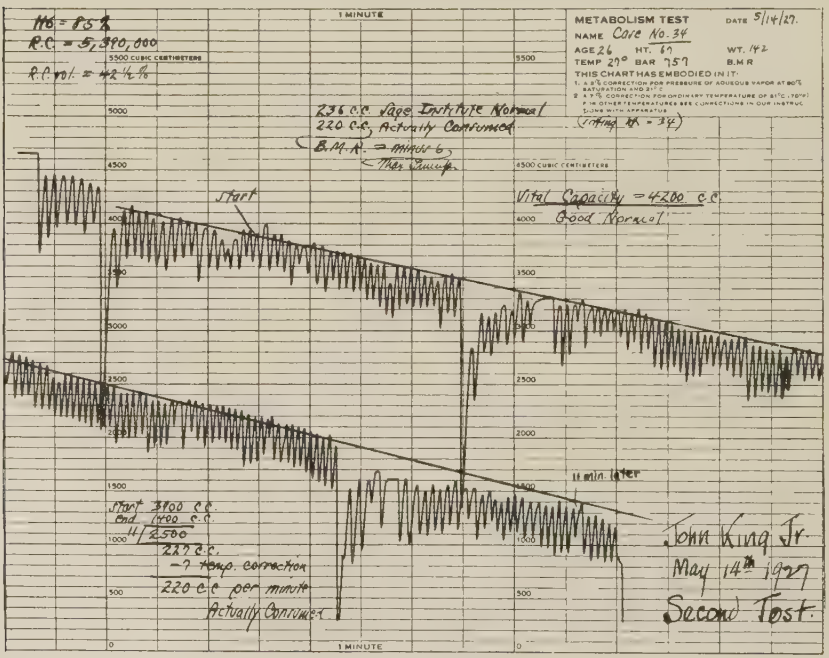
Finally, Group C stands midway between Groups A and B, in volume per breath, dilution factor, and shows a corresponding intermediate position in its red cells and hemoglobin content.

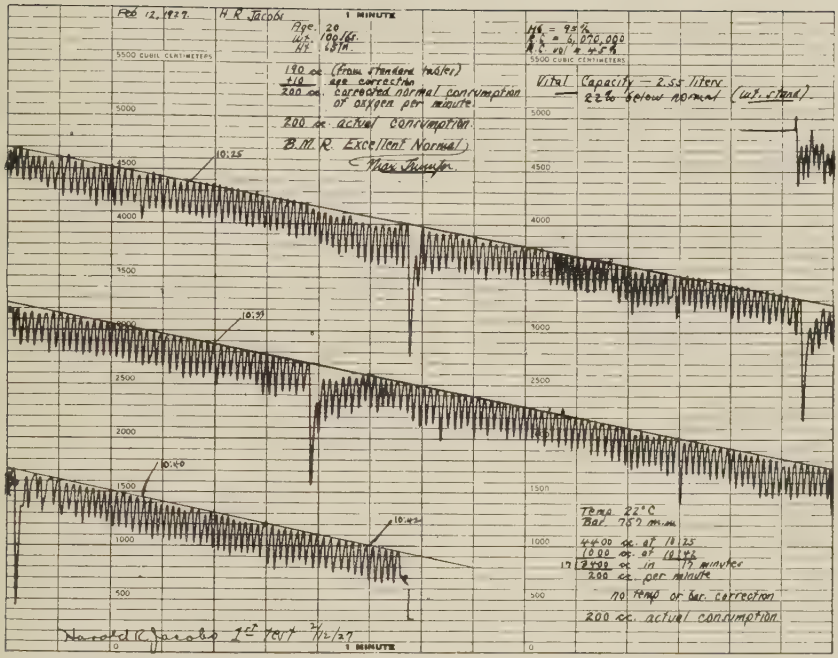
Another test of my theory lies in the dilution factor. This represents the relation of the oxygen consumed per minute to the total ventilation per minute. For example, if an individual consumes 250 cc. of oxygen per minute and his total minute ventilation is 6 liters, then the dilution factor is 24. Thus, in any instance where the total ventilation is not quite sufficient to supply the metabolic demand for oxygen, the dilution factor will be low, with consequent compensatory rise in the hemoglobin and red cells. On examining Group A on page 33, it will be noted that the average dilution factor is 24, the lowest of the three groups, while the average red cell count is 6,224,000, being the highest of the three groups. Thus the high red cell count compensated for the low dilution factor. On examining this group closely, one finds a few individual exceptions—which may be due to over-ventilation caused by hyper-excitability, to faulty circulation or aëration, to low vital capacity or to overweight. Cases 85 and 53 are exceptions, the former is due to overweight and the latter to a low vital capacity.

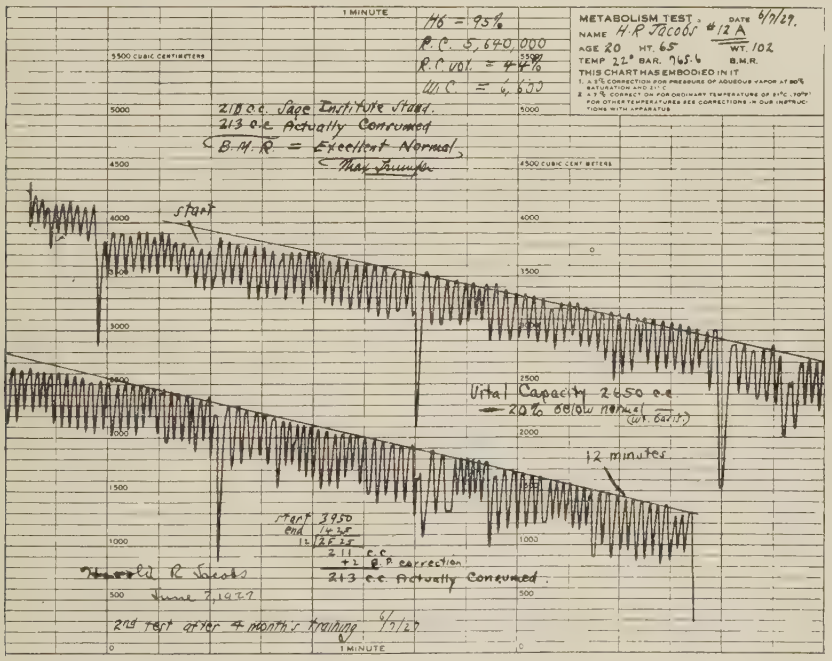
Those cases in which periodic deep breaths were taken in an attempt to assist in the compensation for anoxemia can be studied only from the graphs. (See pages 40 to 42.) The statistical data in these cases do not give a true picture of the manner of breathing. From the charts, it will be seen at a glance that the periodic gasp for air gives a twofold type of breathing—a compensating normal and an occasional deep breath. As previously explained,[1] deep breathing reduces the relative dead space and thereby increases the alveolar or true ventilation. This deep breath also raises the average of the volume per breath above that which the individual is really drawing over 95 per cent of the time.

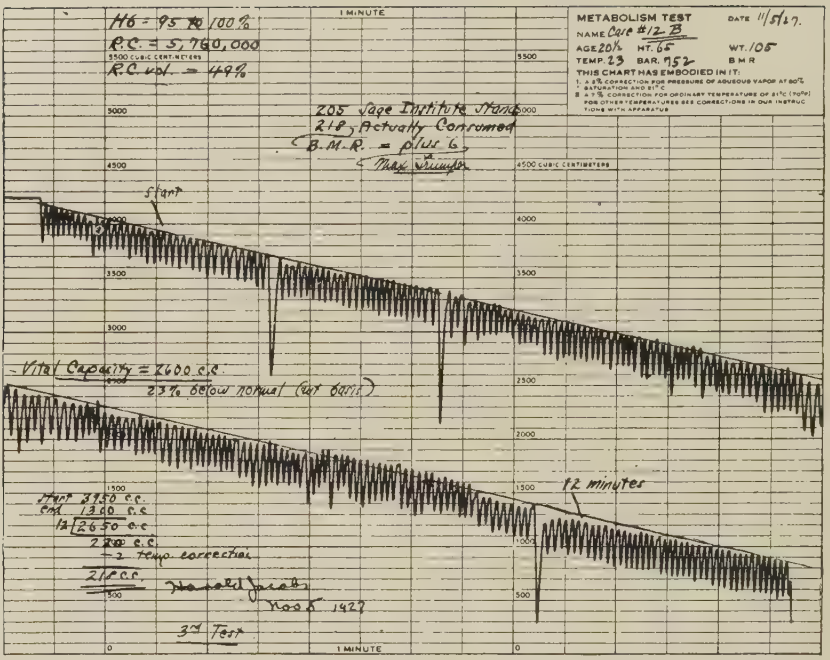
[1] See pages 22 and 23.

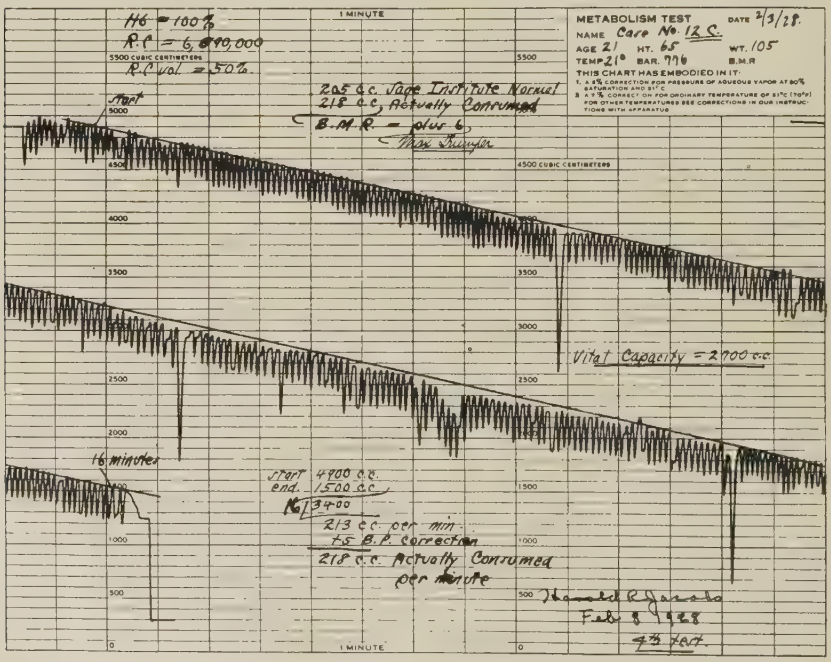

METABOLISM TEST
NAME Case No P3
AGE 13 3/4 HT. 61 1/2 WT. 89
TEMP 73° BAR. 762 B.M.R
THIS CHART HAS EMBODIED IN IT:
1. A 5% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 81° C
2. A 7% CORRECTION FOR ORDINARY TEMPERATURE OF 21° C (70°F) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS
(sitting Ht 30¼)
H6 = 83%
R.C. = 5,520,000
R.C. vol. = 40%
220 c.c. (Sage Institute Normal)
201 c.c. Actually Consumed
(B.M.R. = minus 9
Max Inspir.
Start
1 MINUTE
5500 CUBIC CENTIMETERS
4500 CUBIC CENTIMETERS
V.C. = 3900 c.c.
12% above Normal
14 minutes
Alvin Berg
Dec. 22, 1927
535 Oxford St
Start 4330 c.c.
End 1500 c.c.
14 | 2830
203 c.c.
-2 temp. correction
201 c.c Actually Consumed per minute

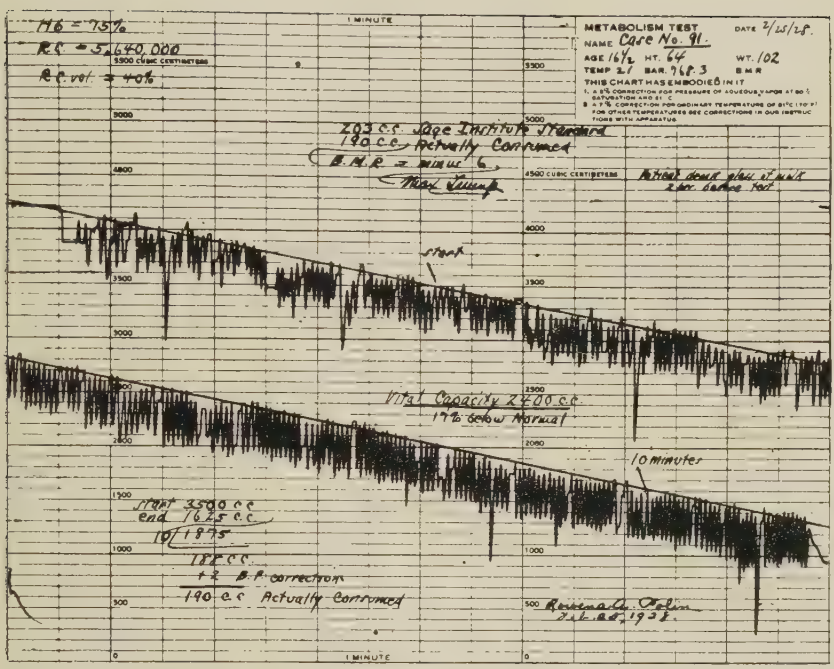

METABOLISM TEST
NAME Case No. 34
AGE 24 HT. 69 WT. 142
TEMP 29° BAR 759 B.M.R
THIS CHART HAS EMBODIED IN IT:
1. A 5% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 81° C
2. A 7% CORRECTION FOR ORDINARY TEMPERATURE OF 21° C (70°F) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS
(sitting Ht = 34)
H6 = 85%
R.C. = 5,390,000
R.C. vol. = 42½%
235 c.c. (Sage Institute Normal)
220 c.c. Actually Consumed
(B.M.R. = minus 6
Max Inspir.
Start
1 MINUTE
5500 CUBIC CENTIMETERS
4500 CUBIC CENTIMETERS
Vital Capacity = 4200 c.c.
Good Normal
11 min later
Start 3100 c.c.
End 1400 c.c.
11 | 2500
227 c.c.
-7 temp. correction
220 c.c per minute
Actually Consumed
John King Jr.
May 14th 1927
Second Test.

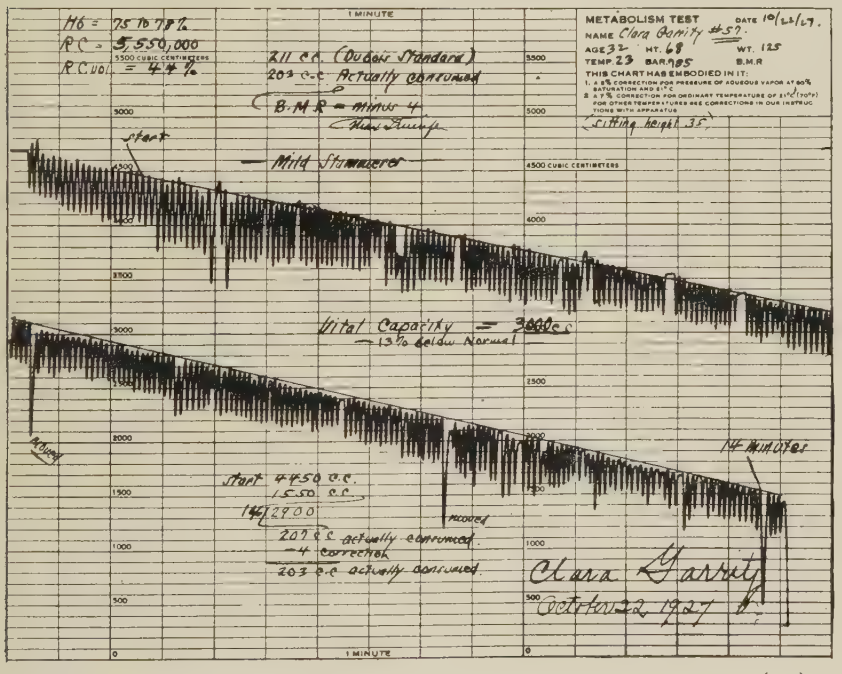

Feb 12, 1927
H R Jacobs
1 MINUTE
Age. 20
Wt. 100 lbs.
Ht. 68 in.
190 cc (From standard tables)
+10 age correction
200 cc. corrected normal consumption of oxygen per minute.
200 cc. actual consumption.
B.M.R. Excellent Normal)
Max Zeman
H6 = 95%
R.C. = 4,070,000
R.C. vol. = 4,458
W.C. = 4,280
Vital Capacity — 2.55 liters
22% below normal (w.s. chart)
5500 CUBIC CENTIMETERS
10:25
10:39
10:50
10:42
Temp 22°C
Bar. 752 mm.
4400 cc. at 10.25
1000 cc. at 10.42
17 3400 cc. in 17 minutes
200 cc. per minute
no temp or bar. correction
200 cc. actual consumption
Harold R Jacobs 1st test 2/12/27
1 MINUTE

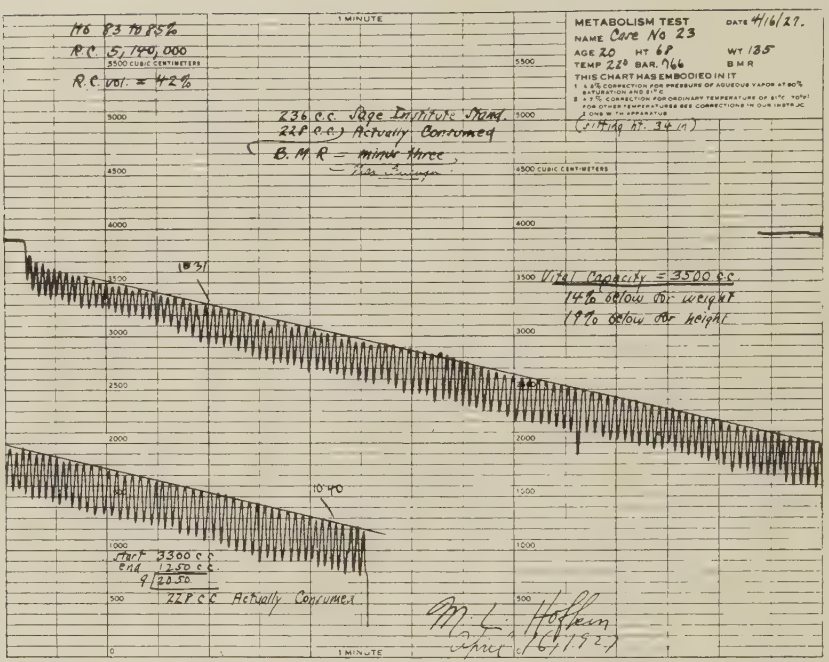

1 MINUTE
H6 = 95%
R.C. 5,640,000
R.C. vol. = 4,418
W.C. = 4,600
210 cc. Sage Institute stated.
213 c.c Actually Consumed
B.M.R = Excellent Normal,
Max Zeman
5500 CUBIC CENTIMETERS
start
Vital Capacity 2650 c.c
— 20% below normal (W.S. Chart)
12 minutes
Start 3950
End 1025
2925 c.c.
+2 B.T.P. correction
213 c.c Actually Consumed.
Harold R Jacobs
June 7, 1927
2nd test after 4 month's training 6/7/27
1 MINUTE
METABOLISM TEST
DATE 6/7/29.
NAME H.R Jacobs #12A
AGE 20 HT. 65 WT. 102
TEMP 22° BAR. 765.6 B.M.R.
THIS CHART HAS EMBODIED IN IT
1. A 3% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 21°C
2. A 3% CORRECT ON FOR ORDINARY TEMPERATURE OF 21°C. (70°F) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS

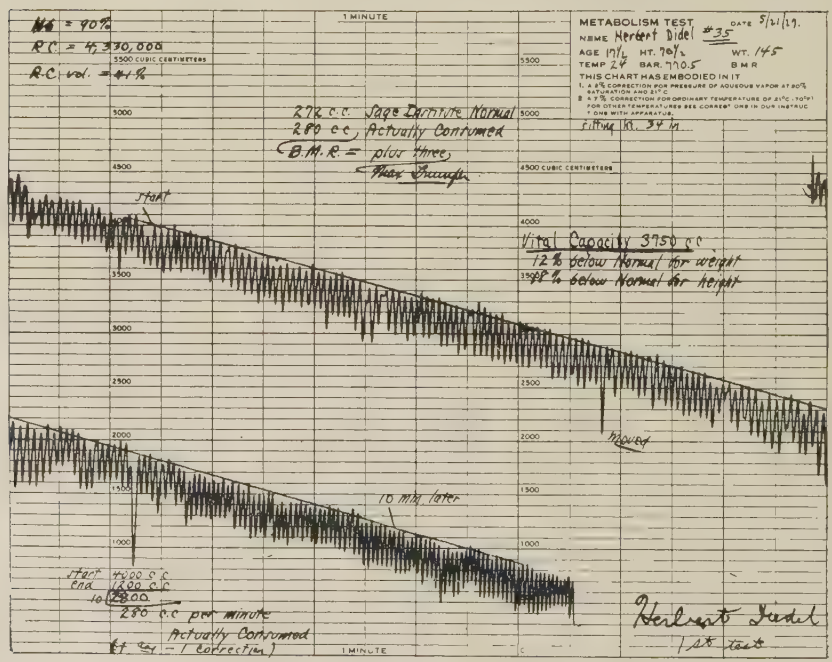

METABOLISM TEST
DATE 11/5/27.
NAME Case #12 B
AGE 20½ HT. 66 WT. 105
TEMP. 23 BAR. 752 B.M.R
THIS CHART HAS EMBODIED IN IT:
1. A 3% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 21° C.
2. A 7% CORRECTION FOR ORDINARY TEMPERATURE OF 21°C (70°F) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS
1 MINUTE
H6 = 95 to 100%
R.C. = 5,760,000
R.C. vol. = 49%
205 Sage Institute Normal
218, Actually Consumed
B.M.R. = plus 6
Max Kriegler
Start
Vital Capacity = 2600 c.c.
23% below normal (cut bottom)
12 minutes
Start 3350 c.c.
End 1300 c.c.
12 | 2650 c.c.
220 c.c.
-2 Temp. correction
218 c.c.
Harold Jacob
Nov 6 1927
3rd Test
5500 CUBIC CENTIMETERS
4500 CUBIC CENTIMETERS


METABOLISM TEST
DATE 2/3/28.
NAME Case No. 12 S.
AGE 21 HT. 65 WT. 105
TEMP 21° BAR. 776 B.M.R
THIS CHART HAS EMBODIED IN IT:
1. A 3% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 21° C.
2. A 7% CORRECTION FOR ORDINARY TEMPERATURE OF 21°C (70°F) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS
1 MINUTE
H6 = 100%
R.C. = 6,890,000
R.C. vol. = 50%
205 c.c. Sage Institute Normal
218 c.c. Actually Consumed
B.M.R. = plus 6
Max Kriegler
Start
Vital Capacity = 2700 c.c.
16 minutes
Start 4900 c.c.
end. 1500 c.c.
№ 3400
213 c.c. per min.
+5 B.P. correction
218 c.c. Actually Consumed per minute
Harold R. Jacobs
Feb 3 1928
4th Test
5500 CUBIC CENTIMETERS
4500 CUBIC CENTIMETERS


METABOLISM TEST
NAME Case No. 91.
AGE 16½ HT. 64 WT. 102
TEMP 21 BAR. 968.3 B.M.R
THIS CHART HAS EMBODIED IN IT
1. A 3% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 21° C.
2. A 7% CORRECTION FOR ORDINARY TEMPERATURE OF 21° C (70° F.) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS
M6 = 75%
R.C. = 5,640,000
R.C. vol. = 406
1 MINUTE
203 c.c. Sage Institute Standard
190 c.c. Actually Consumed
B.M.R. = minus 6
Mean Slump
Patient could glass at mark 2 hr. before test
start
Vital Capacity 2700 c.c.
17% below Normal
10 minutes
start 3500 c.c.
end 1625 c.c.
10/1875
188 c.c.
+2 B.P. correction
190 c.c. Actually Consumed
Reasonably Calm
Jul. 28, 1927.
1 MINUTE


METABOLISM TEST
NAME Clara Garrity #52.
AGE 32 HT. 68 WT. 125
TEMP 23 BAR. 985 B.M.R
THIS CHART HAS EMBODIED IN IT
1. A 3% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 21° C.
2. A 7% CORRECTION FOR ORDINARY TEMPERATURE OF 21° C (70° F.) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS
(Sitting height 35")
M6 = 75 To 79%
R.C. = 5,550,000
R.C. vol. = 4.4%
1 MINUTE
211 c.c. (Outair Standard)
203 c.c. Actually consumed
B.M.R. = minus 4
Mean Slump
start
Mild Shiverer
Vital Capacity = 3000 c.c.
—13% below Normal
14 minutes
start 4450 c.c.
1550 c.c.
1452900
recorded
207 c.c. actually consumed
—4 correction
203 c.c. actually consumed
Clara Garrity
October 22, 1927
1 MINUTE


METABOLISM TEST
NAME Case No 23
AGE 20 HT 68 WT 135
TEMP 22° BAR. 746 B M R
THIS CHART HAS EMBODIED IN IT
1 A 4% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 21°C
2 A 3% CORRECTION FOR ORDINARY TEMPERATURE OF 21°C. (70°F) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUC TONS W TH APPARATUS
DATE 4/16/27.
H6 83 to 85%
R.C. 5,140,000
R.C. vol. = 42%
236 c.c. Sage Dilution Normal
228 c.c. Actually Consumed
B. M. R = minus three
Miss Swingle
Sitting Ht. 34 in
Vital Capacity = 3500 c.c.
14% below Nor. weight
19% below Nor height
10 40
Start 3300 c.c.
End 1250 c.c.
9 | 20.50.
228 c.c Actually Consumed
M. L. Hoffman
April 16/1927
1 MINUTE
3500 CUBIC CENTIMETERS
4500 CUBIC CENTIMETERS


METABOLISM TEST
NAME Herbert Didel #35
AGE 19½ HT. 70½ WT. 145
TEMP 24 BAR. 730.5 B M R
THIS CHART HAS EMBODIED IN IT
1 A 4% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 21°C
2 A 3% CORRECTION FOR ORDINARY TEMPERATURE OF 21°C. (70°F) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUC TIONS WITH APPARATUS.
DATE 5/21/29.
H6 = 90%
R.C. = 4,330,000
R.C. vol. = 41%
272 c.c. Sage Dilution Normal
280 c.c. Actually Consumed
B.M.R. = plus three
Miss Swingle
Sitting Ht. 34 in
Start
Vital Capacity 3750 c.c
12% below Normal for weight
3% below Normal for height
moved
10 min. later
Start 4200 c.c
End 1300 c.c
10 | 28.00
280 c.c per minute
Actually Consumed
(+ 3% - 1 correction)
Herbert Didel
1st test
1 MINUTE
3500 CUBIC CENTIMETERS
4500 CUBIC CENTIMETERS

However, the outstanding point of interest in these cases is the constancy of the curves obtained in the same individual at various times. Pages 41, 42 show 4 graphs on the same patient during a period of one year. Incidentally, the second test was made after he had completed four and a half months' speech training and the breathing curve demonstrates his improvement. Subsequently, he has relapsed and on the third and fourth tests his breathing and compensating hematologic functions are no better than they were the year before when the first test was made.

There were 11 cases having an average vital capacity 13 per cent below normal and they compensated either by an increase in the depth of respiration, or by an increased hemoglobin content and erythrocyte count or by both. (See graphs on page 44.) There were also 3 cases having an average vital capacity of 27 per cent below normal and they compensated by a still greater depth of respiration. Graph on page 9 illustrates this, in case No. 90.

There were 10 cases where the depth of respiration was either normal or above, with the hemoglobin and erythrocyte count also normal. These are illustrated on page 53.

It is recognized that stammerers commonly suffer from nose and throat affections.[1] R. M. Lukens[2] examined about one-third of the cases under consideration, and made the following report:

"Thirty-two cases of stammerers were examined and ranged in age from fifteen to twenty-seven years. There were 28 males and 4 females.

"*Nasal Findings.*—Only 3 of the 32 cases had normal noses. Twenty-nine cases had rhinitis in varying degrees of severity, or obstructive deformities of the turbinates or septum or both.

"*Pharyngeal Findings.*—Six cases had previous tonsillectomies with more or less satisfactory results, excepting that some of them had damaged pillars and in one, the tonsillectomy was incomplete. Twelve of the cases had infected tonsils, some of

[1] Makuen (1914) in his extensive studies on stammerers reports over 37 per cent had intranasal pressure and about 60 per cent had diseased tonsils and adenoids.

[2] Personal communication of unpublished data from Dr. R. M. Lukens, Laryngologist to the Chest Department, Jefferson Hospital.

which were small, some were hypertrophied and some were submerged. Most of the cases had chronic pharyngeal inflammation.

"*Laryngeal Findings.*—Nine cases had normal larynges. Twenty-one had laryngitis in varying degrees. In 2 cases there was marked fluttering of the vocal cords on deep inspiration. In one case there was spasm of the arytenoideus muscle. The arytenoids, when first seen on quiet respiration, were separated and in the normal abducted position for respiration. Adduction of the vocal cords and arytenoid cartilages during phonation was normal, but on inspiration following phonation the vocal processes of the arytenoid cartilages were rotated outward but the cartilages themselves were held together by spasm of the arytenoideus muscle. Three cases showed infantile epiglotti.

"Of 15 cases tested for anesthesia of the larynx, 4 cases apparently were anesthetic to the application of a dry cotton-tipped probe. All the other cases promptly coughed with this test."

Of the total group of stammerers, there were 4 cases with marked nasal obstruction. Case No. 16 is an excellent example of a stammerer who has developed a tolerance for carbon dioxide and his graph on page 19 shows this clearly. The graph of Case No. 25 shows the normal response. The metabolic curve of Case No. 16 is shown on page 48. Dr. Starr found his alveolar carbon dioxide to be 6.9 per cent. The respiratory rate is normal, but the volume per breath and the minute ventilation are both increased. His vital capacity is normal. The dilution factor of oxygen is markedly increased to 34. This bears out my hypothesis, for his hemoglobin is 90 per cent, red cells 5,100,000. In other words, the blood picture is normal[1] except that his white count is 12,200, but his carbon dioxide tolerance is high along with his alveolar CO_2. This was a *case with nasal obstruction.* Later this obstruction was removed by a surgeon in the University of Pennsylvania Hospital. After one month's speech training, he had completely recovered from his stammering.

[1] The differential count was:

Polymorphonuclears	74 per cent	Large lymphocytes	2 per cent
Small lymphocytes	20 per cent	Transitionals	3 per cent
		Basophile	1 per cent

This case is strikingly similar to one reported by Makuen[1] in 1896 of a child who suddenly ceased to stammer after the removal of adenoids. Dr. Makuen stated: "I thought I had made a discovery but further experience has led me to believe that the good result obtained in that single case was unusual and in the nature of a coincidence."

I am inclined to believe that Dr. Makuen had really made a discovery and that the good result which he had obtained was not a coincidence but the consequence of the removal of an obstruction which had been causing a chronic form of respiratory fatigue.[2] In conditions such as adenoids, the resulting fatigue causes the carbon dioxide to find its exit by channels other than the respiration.[3] Starr, in his pioneer studies on salivary pH and alveolar carbon dioxide in this laboratory[4] found that sub-breathing stammerers are overloaded with carbon dioxide far in excess of the normal individual. He further found in normal individuals the pH of the mixed saliva varied directly with the carbon-dioxide content of the alveolar air, thus indicating that sub-breathing stammerers would show a high alveolar carbon-dioxide content which he subsequently found to be the case. Friedman and Jackson (1917) found high carbon-dioxide content of the alveolar air in obstructed respiration.

There were three other cases with nasal obstructions:[5]

[1] Makuen, G. H.: Study of 1000 cases of Stammering, etc., Trans. Coll. Phys., 1914, **36**, 110.

[2] Davies, Priestly and Haldane, Jour. Physiol., 1919, **53**, 57, studied fatigue of respiration and found men with symptoms of neurasthenia particularly sensitive to respiratory resistance.

[3] McDowell's Clinical Physiology (1927), page 149.

[4] Psycho-Biochemistry Laboratory of the Speech Clinic of the Graduate School of the University of Pennsylvania.

[5] *Case No.* **99.** Seventeen years. 140 pounds.

Volume per breath	392 cc.	Vital capacity (−16)	= 3400 cc.
Respiratory rate	16	Hemoglobin	98 per cent
Dilution factor	26	Red cells	5,380,000

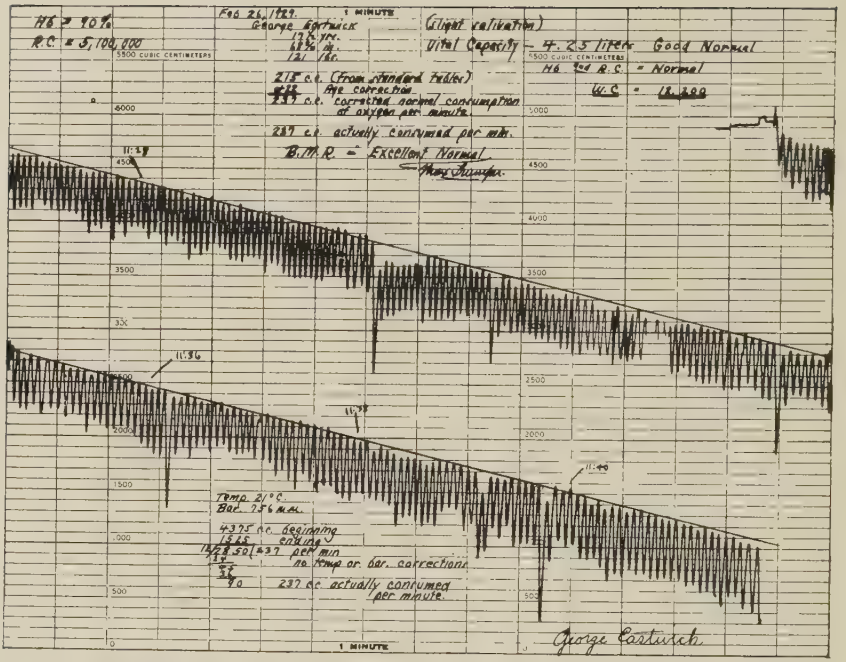
Case No. **86.** Eighteen years. 140 pounds.

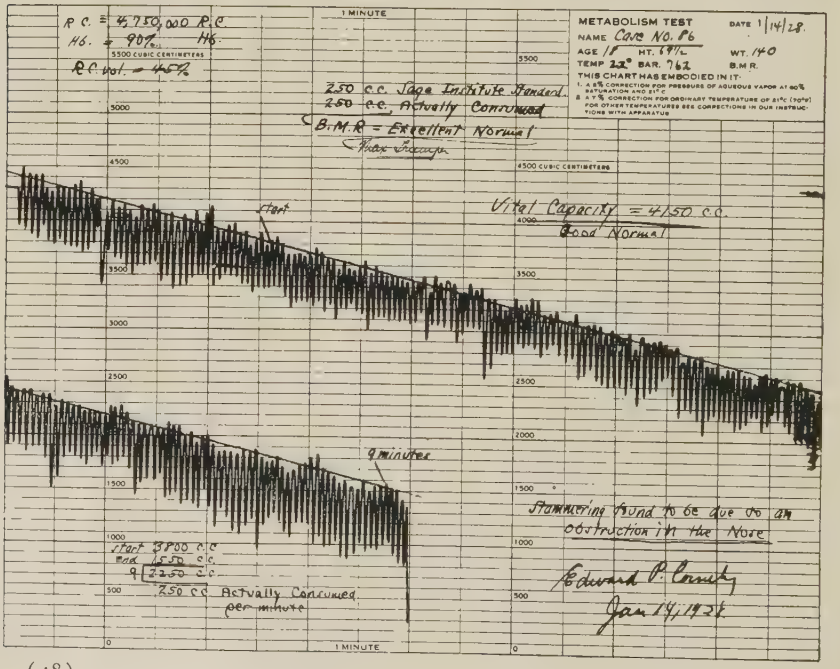
Volume per breath	511 cc.	Vital capacity (N)	= 4150 cc.
Respiratory rate	18	Hemoglobin	90 per cent
Dilution factor	37	Red cells	4,750,000

Case No. **82.** Nineteen years. 123 pounds.

Volume per breath	410 cc.	Vital capacity (+10)	= 4200 cc.
Respiratory rate	14	Hemoglobin	95 per cent
Dilution factor	25	Red cells	5,950,000

These 3 patients promised to have the obstruction removed.


Feb 26/1929
George Eastwick
(Slight exhilaration)
Vital Capacity - 4.25 liters Good Normal
H6 for R.C. = Normal
W.C. = 12,200
215 c.c. (From standard tables)
Age correction
237 c.c. corrected normal consumption of oxygen per minute.
237 c.c. actually consumed per min.
B.M.R. = Excellent Normal
Harry Benedict
11:34
11:36
11:39
11:40
Temp. 21°C.
Bar. 736 m.m.
4375 c.c. beginning
4137 ending
238 c.c. 50 237 per min
no temp or bar. corrections
237 cc actually consumed per minute.
George Eastwick
1 MINUTE
5500 CUBIC CENTIMETERS


R.C. = 4,750,000 R.C.
H6. = 90% H6.
R.C. vol. = 45%
250 c.c. Sage Institute Standard
250 c.c. Actually Consumed
B.M.R. = Excellent Normal
Harry Benedict
METABOLISM TEST
DATE 1/14/28.
NAME Case No. 86
AGE 18 HT. 69" WT. 140
TEMP 22° BAR. 762 B.M.R.
THIS CHART HAS EMBODIED IN IT:
1. a 6% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 90% SATURATION AND
2. a 3% CORRECTION FOR ORDINARY TEMPERATURE OF 21°C (70°F)
FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS
start
Vital Capacity = 4150 c.c.
Good Normal
9 minutes
Stammering found to be due to an obstruction in the Nose
Edward P. Connely
Jan 14, 1928
Start 3900 c.c.
End 1650 c.c.
9 | 2250 c.c.
250 cc Actually Consumed per minute
1 MINUTE
5500 CUBIC CENTIMETERS

Case No. 82 was the most severe stammerer of the four. He had congestion of nasal mucous membrane, with hypertrophy of the middle turbinates. His dilution factor was normal, and since he did not compensate by increased volume of breathing— compensation followed by increase of hemoglobin and red cells.

None of the cases with nasal obstruction were shallow breathers. On the contrary, the nasal obstruction causes a slight resistance to the free flow of air in and out of the lungs and tends to induce a slower and deeper form of respiration. Cases 16 and 86 compensated for the obstruction by a definite increase in the volume per breath and the total ventilation as evidenced by the high dilution factors of 34 and 37, with normal hemoglobin and red count. Case 99 did not compensate by increased breathing as evidenced by the fact that his dilution factor was normal, namely 26, and so he compensated by a hemoglobin of 98 per cent and a slight increase in red cells.

COMPENSATION AS AFFECTED BY VITAL CAPACITY.

Practically one-half of the adult stammerers had lowered vital capacities.[1] There were 32 cases with a vital capacity between 10 per cent and 38 per cent *below normal.*

One of the factors that reduce vital capacity mentioned by Moersch (1925) is defect of posture. A large percentage of these cases had postural defects, their shoulders drag forward and downward and this prevents the lungs from expanding as they normally should. Myers (1925) in his summary of the influence of disease on the vital capacity concludes that when it is 15 per cent or more below the normal, the subject should be most carefully studied for disease conditions interfering directly or indirectly with the functions of the heart and lungs. We are unable at present to determine the causes of the rather low vital capacity in non-pathologic conditions. Whether it is possibly the result of disuse, as in stooped shoulders and in shallow breathing, or to nervous factors as in vagatonia is not known with certainty. But in any case this does not detract from the clinical significance of my findings.

The salient feature of these findings is that individuals with a low vital capacity compensated in one of the following ways:

1. Normal breathing broken at intervals by an extra deep breath. See graphs on pages 40–42 (11 showed this characteristic).

2. Rapid shallow breathing typical of anoxemia. See graphs on page 52 (6 showed this characteristic).

3. Definite increase in the volume per breath. See graphs on page 43 (14 showed this characteristic).

Of the 26 adult cases with normal vital capacity, 12 were shallow breathers.

Of the 16 adult cases with vital capacity averaging 9 per cent above normal, eight were shallow breathers.

It is seen that in all three groups, namely, those having low, normal and high vital capacity, from 15 per cent to 50 per cent were shallow breathers.

[1] Of 82 adult stammerers, 40 had a low vital capacity. That the frequency of the low vital capacity was not due to fatigue of the respiratory muscles is apparent from the work of Peabody and Sturgis (1917) on patients suffering from pernicious anemia and cardiac disease. Tests were made every fifteen seconds for ten minutes and there was no difference in vital capacity at the end of ten minutes.

Thus of the three groups mentioned above, showing respectively low, normal and high vital capacity, the last has the highest frequency of shallow breathing, namely, 50 per cent. Next is the group with normal vital capacity showing a shallow breathing frequency of 46 per cent. Finally, the group having vital capacity below normal has the lowest frequency of shallow breathing, namely 15 per cent. This is directly at variance with the statement made by Professor Means of the Harvard Medical School[1] that "the reason for the shallow type of breathing lies chiefly in a restriction in the vital capacity, although according to the theories[2] of the Oxford School, it may in part be the result of anoxemia, for not only, so Haldane, Meakins and Priestly tell us, does rapid shallow breathing produce anoxemia but the hyperpnea induced by anoxemia often is of a rapid shallow type. Thus a vicious circle is established. This, however, is all pretty theoretical whereas the restriction in vital capacity is a well established fact." I found, on the contrary, that *individuals with low vital capacity are the least likely to be shallow breathers. In fact, the smaller the vital capacity, the less likelihood there is for them to be shallow breathers.* Eleven of my cases[3] having an average vital capacity of 13 per cent below normal compensated by an increase in the depth of respiration. Their volume per breath average 490 cc., which is equivalent to 15.2 per cent of their vital capacity. Whereas, authorities agree that the volume per breath normally is 10 to 12 per cent of the vital capacity. Besides the above eleven, there were 3 cases[4] with still smaller vital capacity, averaging 27 per cent below normal. These extreme cases showed an average volume per breath of 603 cc., which is equivalent to 19 per cent of their vital capacity. (See graph of Case No. 90 on page 9.)

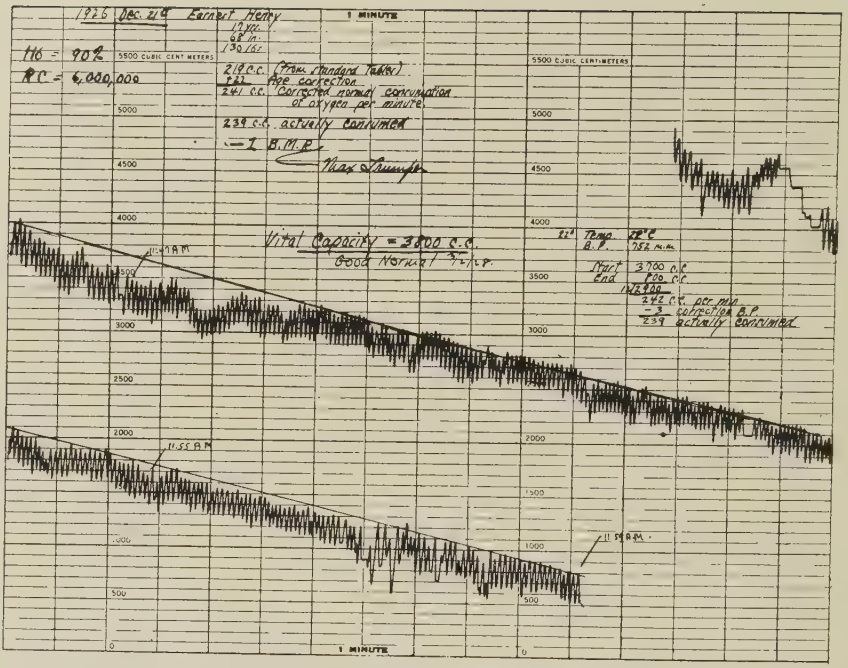
My findings substantiate the views of the Oxford School, namely, that oxygen want prevails when the lungs expand unevenly due to rapid shallow breathing.

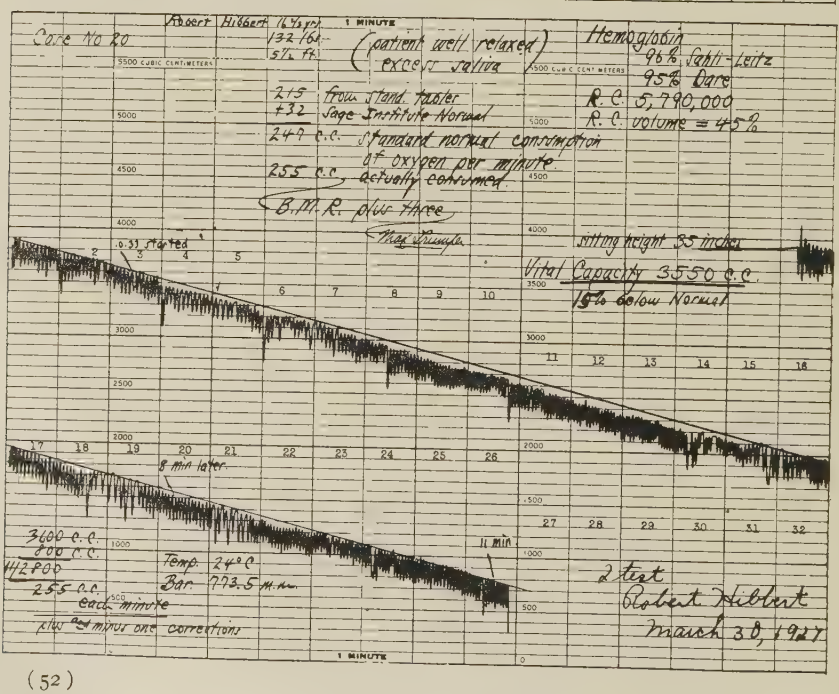
[1] Monograph on Dyspnea, 1924, p. 86.

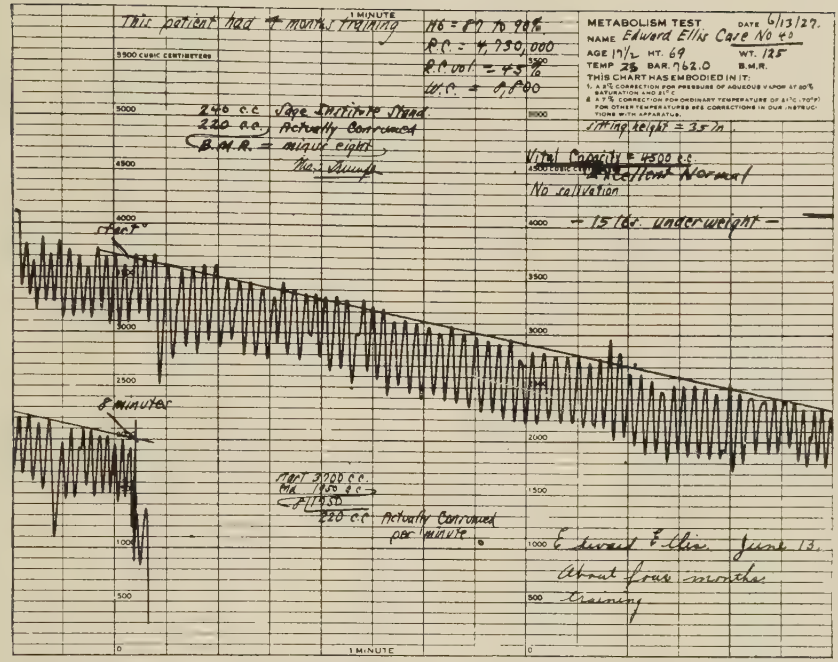
[2] "In brief, Haldane, Meakins and Priestly believe that rapid shallow breathing tends to produce anoxemia; this being due, they think, to an incomplete expansion of the lungs and a passage of a portion of the blood through lung areas where it is incompletely oxidized."

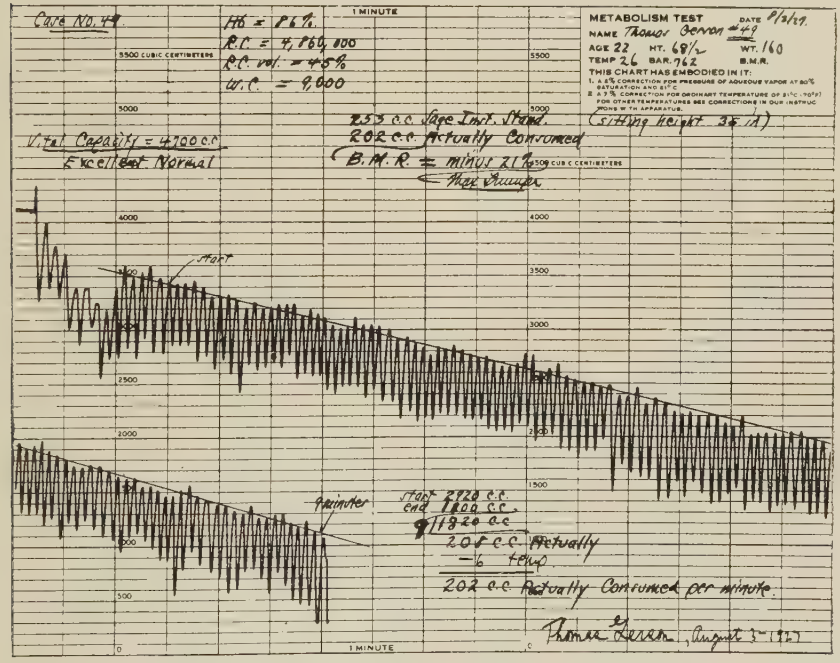
[3] Average respiratory rate 15.5; 131 pounds, and twenty-one years.

[4] Average respiratory rate 11.0; 146 pounds, and twenty-four years.


1925 Dec. 21 Earnest Henry
1 MINUTE
Hb = 90%
RC = 6,000,000
5500 CUBIC CENT METERS
219 c.c. (From standard Table)
+22 Age correction
241 c.c. Corrected normal consumption of oxygen per minute.
239 c.c. actually consumed
—2 B.M.R.
Max Stumpf
Vital Capacity = 3800 c.c.
Good Normal S.T.&P.
11:47 AM
11:55 AM
Temp. 72°C
B.P. 758 m.m.
Start 3700 c.c.
End 3700 c.c.
1/2 400
342 c.c. per min
-3 correction B.P.
239 actually consumed


Case No 20
Robert Hibbert 16½ yrs.
132 lbs.
5 ft. 7½ in.
1 MINUTE
5500 CUBIC CENTIMETERS
(patient well relaxed) except saliva
215
+32 From stand. tables Sage Institute Normal
247 c.c. Standard normal consumption of oxygen per minute
255 c.c. actually consumed
B.M.R. plus three
Max Stumpf
Hemoglobin 96% Sahli-Leitz 95% Bare
R.C. 5,790,000
R.C. volume = 45%
0.31 started
sitting height 35 inches
Vital Capacity 3550 c.c.
15% below Normal
8 min later
11 min
3600 c.c.
200 c.c.
412 800
255 c.c. each minute
plus and minus one corrections
Temp. 24° C
Bar. 723.5 m.m.
2 test
Robert Hibbert
March 30, 1927


This patient had 4 months training
H6 = 99 to 98%
R.C. = 4,750,000
R.C. vol. = 95%
W.C. = 9,500
METABOLISM TEST
DATE 6/13/29.
NAME Edward Ellis Case No 40
AGE 19½ HT. 69 WT. 125
TEMP 25 BAR. 762.0 B.M.R.
THIS CHART HAS EMBODIED IN IT:
1. a 4% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 21°C.
2. a 7% CORRECTION FOR ORDINARY TEMPERATURE OF 21°C. (70°F) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS.
Sitting Height = 35 in.
240 c.c. Sage Institute Stand.
220 c.c. Actually Consumed
B.M.R. = minus eight
No. Thump
Vital Capacity = 4500 c.c.
Excellent Normal
No salivation
— 15 lbs. underweight —
Start
1 minute
Start 3000 c.c.
End 1780 c.c.
1220 c.c. Actually Consumed per minute
E. Ellis June 13.
About four months
training


Case No. 44
H6 = 96%
R.C. = 4,760,800
R.C. vol. = 95%
W.C. = 9,600
METABOLISM TEST
DATE 9/2/29.
NAME Thomas Gerron #49
AGE 22 HT. 68½ WT. 160
TEMP 26 BAR. 762 B.M.R.
THIS CHART HAS EMBODIED IN IT:
1. a 4% CORRECTION FOR PRESSURE OF AQUEOUS VAPOR AT 80% SATURATION AND 21°C.
2. a 7% CORRECTION FOR ORDINARY TEMPERATURE OF 21°C. (70°F) FOR OTHER TEMPERATURES SEE CORRECTIONS IN OUR INSTRUCTIONS WITH APPARATUS.
(Sitting Height 36 in)
Vital Capacity = 4700 c.c.
Excellent Normal
253 c.c. Sage Inst. Stand.
202 c.c. Actually Consumed
B.M.R. = minus 21%
Start
1 minute
Start 2220 c.c.
End 1800 c.c.
420 c.c.
20 c.c. Actually
— ½ temp
202 c.c. Actually Consumed per minute.
Thomas Gerron, August 3-1927

SHALLOW BREATHING.

The shallow breather is comparable to the individual ascending to high altitude, both being subject to the same physiologic response to the anoxemia condition. One of the most striking features of this response is an increase in hemoglobin and red cells compensating for the diminution in arterial oxygen tension. The stammerers who are shallow breathers become, in this respect, acclimated to their chronic condition just as the mountain climber does in the more acute conditions of high altitude. The degree of erythrocytosis which is produced in normal persons subjected at high altitudes to a low oxygen tension is variable because it is an acute condition and also because of the individual variations in pulmonary permeability and in the sensitiveness of the respiratory center. Schneider and Truesdell (1922) found in their studies of anoxemia that individuals differ in their capacity to respond to and endure low oxygen. These differences were clearly brought out with anoxemia that was developed slowly. Harrop and Heath (1927) are of a similar opinion. This, then, should account for the fact that in my series of unpicked cases there were some exceptions. In fact, it is surprising that there were so few exceptions, and I am inclined to believe that in those stammerers with mild erythrocytosis, the condition has been present for such a long time that the abnormal rhythm becomes for a given individual a fixed type of breathing. A good illustration of this is Case No. 12 (pages 41, 42), showing spirographic curves covering one year. We know, for instance, that workers exposed daily to small amounts of carbon monoxide[1] show a mild erythrocytosis due to an anoxemia, because the oxygen capacity of the blood is diminished by whatever amount of hemoglobin is combined with carbon monoxide. Other conditions causing anoxemia with resulting shallow breathing and increased hemoglobin and red cells are reported by many workers. Hunt and Price-Jones

[1] Haines, Karasek, Apfelback Report, p. 193, Trumper's Memoranda of Toxicology.

(1918) first indicated the occurrence of polycythemia in chronic gas poisoning. Of the 73 cases in which they enumerated the red cells, the highest count was 6,900,000 with a hemoglobin of 118 per cent. Barcroft and his co-workers (1918) also mention a high red count in chronic gassed cases. Dorothy Dufton (1918) endeavored to reproduce experimentally the gassing of soldiers by exposing rabbits and goats to similar concentrations of chlorine and phosgene and concluded from her experiments that chronic polycythemia is caused by oxygen want. Underhill (1926) while contending that it is almost impossible to assign a cause or attempt to correlate the symptoms of gas poisoning, emphasizes the fact that the one constant finding is change in blood concentration. Surgeon-General Ireland (1926) states that the sequel of gas poisoning is shallow breathing and that these patients are often polycythemic. He also mentions that various nervous manifestations are commonly found but are not associated with any organic lesion. This resembles not only the hemato-respiratory picture frequently found in stammering but also its nervous manifestations which likewise are not associated with any organic lesion. Harrop and Heath (1927) in their excellent studies on polycythemia vera, mention that the nervous symptoms are among the most common and it is these symptoms which are common in mild grades of chronic anoxemia. Of the 7 cases which they reported, 3 had poor memory and 2 *had thickness of speech*. This again has some resemblance to the mild chronic anoxemia found in stammerers.

Since one-third of my adult cases are shallow breathers, I will review briefly some of the conditions in which shallow breathing is common. Stammerers often show a history of an infectious disease like pneumonia. Edsall (1913) mentions severe pulmonary conditions, infectious diseases, simple neurosis and also severe cardiac conditions. Douglas (1917) Haldane and his co-workers (1918) showed this type of breathing to be present in effort syndrome[1] or disordered action of the heart following some of

[1] The "soldier's heart" or "the irritable heart" was first described by J. M. Da Costa of the Jefferson Medical College in 1871. The British at first used the term disordered action of the heart, or D. A. H., and Lewis suggested effort syndrome.

the cases of gas poisoning. Mudd (1925) demonstrated this type of breathing in effort syndrome and also in a case of psychoneurosis of the compulsive type. Means (1924) contends that shallow breathing in pneumonia may be due mechanically to a lowered vital capacity, although he admits that nervous influence may play an important part. Mudd (1925) mentions among the factors that produce the rapid shallow breathing and anoxemia, fatigue of the respiratory center and pleural pain. Wells[1] (1927) found in fatal cases of lobar pneumonia, a waxy degeneration of the fibers of the muscles of respiration, especially the diaphragm. The reason for shallow breathing in all the conditions enumerated may be chemical and it may be nervous. The Oxford School has demonstrated the former, while, in connection with stammering, Stern (1925) in Germany and Gordy (1927) in this country have demonstrated the latter. Dr. Samuel T. Gordy examined a group of my cases and made the following report:[2]

"Routine physical examinations of a group of 25 stammerers discloses certain constitutional factors in the make-up of these individuals, factors which recur with sufficient frequency to arrest attention. The number of cases is not sufficiently great to draw statistical conclusions from any figures based upon these preliminary studies. The conclusions drawn, thus far, are simply clinical impressions.

"The constitutional factors appear to be both structural and functional. The anatomic and bodily contour of the majority of this group belongs to the asthenic type (13 cases). In addition, there are 7 who belonged to the athletic type, 3 to the asthenic-athletic type and 2 cases of the pyknic type.[3] The greater number of cases seem to belong to the visceroptotic group with slouchy and defective posture. The only outstanding endocrine disturbance was of the juvenile hyper-pituitary type with comparative increase in the long bones, large hands and feet and

[1] H. G. Wells (1909) had shown that whenever over-production, under-elimination or under-oxidation occurs, typical waxy degeneration of muscle fibers may ensue.

[2] Personal communication of unpublished data from S. T. Gordy of the Neurological staff of the Jefferson Medical College Hospital.

[3] Kretschmer's Classification (Physique and Character), English translation (London, 1925).

history of very rapid pubertal and adolescent growth (5 cases). There were 2 cases of hypo-pituitarism with distinct tendency to the Froelich syndrome.

"The functional factors are chiefly referable to the vegetative nervous system and are preponderantly those clinically termed vagotonic, which may be defined as an increased activity or irritability of the autonomic (extended vagus) portion of this system. The phenomena occurring frequently are dilated pupils, bradycardia, (heart rate below 60 per minute), cyanosis of extremities frequently with attendant excessive perspiration, diminution of gag reflex (pharyngeal anesthesia), sinus arrhythmia of the heart, visceroptosis (which is a combined structural and functional condition), a few instances of dermatographism and finally subnormal breathing, which of course, is one of the major points in your investigation.

"My uncompleted investigation confirms in very great measure the work of Hugo Stern,[1] who found the same association of many vagotonic phenomena in the majority of stammerers which he examined. Not all of his cases show these vagotonic symptoms, nor do those that have them show all the classical symptoms of vagotonia, described by Eppinger and Hess, but frequently, three, four or five or more of these are shown in one individual and often to a very marked degree.

"The similarity of these types of individuals with their concomitant vagotonia to those individuals who show what is known as the effort syndrome (neurocirculatory asthenia) is striking. Many cases of stammering and many cases of neurocirculatory asthenia have a paralleling of similar psychic and physical factors. In the stammering cases respiratory phenomena appear to predominate whereas those with the effort syndrome seem to have more outstanding circulatory phenomena. In both instances we have physiologic evidence of a disturbed function of a vagus, but with emphasis upon different functions of the vagus system in different types. The psychic counterpart is, of course, present and it appears that both are equally important in the causation and the production of clinical symptoms."

[1] Zur Pathogenese der Dysarthria Spastica, Zeitschr. f. Hals-Nasens- und Ohrenheilkunde, München. und Berlin, 1925, **12**, 278–284; 286–290.

THEORIES OF STAMMERING.

There is an abundance of theories for stammering and a superabundance of methods for treating it. On the other hand there is a dearth of research data. The latter are very much needed for a real understanding and intelligent treatment of this condition. Research findings will also help to expose the many quacks and charlatans who profess sure and short cures by supposedly secret methods. Herewith follow some theories of stammering:

1. Weakness of speech mechanism. (Kustner, 1716.)
2. Spastic neurosis of coördination. (Kussmaul, 1883.)
3. Reduced pressure in pulmonary air. (Cöen, 1883.)
4. Psychoneurosis. (Ssikorski, 1894.)
5. Imitation or heredity.
6. Mental or physical trauma—fear neurosis.
7. Inferiority of cells in auditory area. (Makuen, 1896.)
8. Capacity for words not equal to rich urge for speech.
9. Amnesia, especially prevalent in dextro-sinistrals.
10. Associative aphasia. (Hoepfner.)
11. Inferiority complex. (Adler.) Repressed complexes. (Freud.)
12. Neurosis of compulsion "zwangsneurose." (Trömner.)
13. Vacillation of attention. (Rothe.)
14. Sluggishness of speech reflexes. (Rosenthal.)
15. Transitory auditory amnesia (*caused by cerebral congestion due to fear or fright.* (Bluemel, 1913.)

The fact that stammerers often speak well in solitude, can usually sing and swear without difficulty, can speak well in concert with other persons, can repeat a word after prompting by another person, and can usually repeat words even though they have just been stammering on the same word; all these facts are indications against an organic lesion being the cause of stammering. Further, an explanation of stammering must also involve an interpretation of the fact that congenitally deaf persons never stammer and also of the relative infrequency of stammering in women.

Gutzmann (1912) states that the congenitally deaf never

stammer and this is confirmed by Gallaudet.[1] In learning to speak the deaf person employs sight as a substitute for hearing. He interprets the spoken words of others by reading the lips, tongue and facial muscles. And yet the deaf person is subject to many of the causes usually inducing stammering such as infectious diseases and shock. Because of his deafness, he cannot be induced to stammer through imitation or through the fear of speaking. Blumel contends that the reason for the deaf being free from stammering is that they lack auditory imagery and consequently there cannot be a disturbance of this center to affect their speech.

Ssikorski (1894) in treating of the prevalence of male over female stammerers contends that the motor centers of a woman's left cerebral hemisphere develop, generally speaking, more quickly than a man's and seem less exposed to injuries. This accounts not only for the earlier development of speech with girls, but also for other psycho-motor specialities of women, *viz.*, their skill in dancing, singing and needlework. German statistics show that at least two-thirds of the stammering school girls on reaching adult life have recovered. Greene (1901) found from a study of 256 adult stammerers that the frequency in the male was nine times that of the female. This proportion agrees very closely with that reported by many investigators. In my series the proportion is about eight to one.

Lewis has demonstrated that costal and diaphragmatic breathing have opposite effects upon the blood pressure. He finds that costal inspiration lowers systemic blood pressure while abdominal inspiration raises it. The extent to which interference with diaphragmatic action would affect intra-thoracic pressure depends directly upon the extent to which it interferes with respiration. In women, who tend to be costal breathers such interference would affect respiration less than in men, who are diaphragmatic breathers. Since cerebral venous pressure is dependent to a large degree upon the maintenance of normal intra-thoracic pressure it would naturally follow that cerebral circulation in women would be less subject to interference by abnormalities of diaphrag-

[1] Dr. Gallaudet, President of the Columbia Institute for the Deaf and Dumb, Washington, D. C., states this fact from his experience of fifty years

matic action than in men. Since disturbances of respiration are among the primary factors involved in the production of stammering, this condition would tend to be less frequent in women than in men. The abnormal movements of the breathing muscles usually appear as a tonic or clonic contraction—more pronounced in the movement of the diaphragm than in the movement of the ribs. The normal anachronism between chest and abdominal breathing is disturbed. The undue effort made by the stammerer contributes to the cerebral congestion which has already been set up by the fear of speaking.[1] According to Bluemel (1913) this causes a transitory auditory amnesia which prevents normal speech.

In addition to costal breathing, one of the factors which may account for the scarcity of women stammerers is the lower viscosity of their blood. Viscosity depends principally on the total volume of corpuscles per unit volume of fluid.[2] The normal red cell count and the red cell volume for women is 10 per cent below that of man. Therefore under the increased pressure of abdominal breathing the greater viscosity of man's blood facilitates cerebral congestion since the brain is enclosed in a non-yielding capsule and cannot be enlarged like other organs.

Haldane and Mavrogordato also demonstrated clearly the effects of interruption of natural breathing during each of its phases. When there is a sudden interruption of expiration a marked increase of intrapulmonary and intrathoracic pressure follows. This increased pressure is at first slight and constant, but soon rises rapidly, culminating finally in a prolonged increased inspiratory effort. Of special significance is the effect of these pressure changes upon the cerebral circulation. The marked and prolonged increase in intra-thoracic pressure which accompanies the simple interruption of expiration is greatly augmented by the act of stammering, during which it is apparent that several forced respiratory efforts are made with the glottis in clonic spasm. This latter phenomenon may well be a manifestation of

[1] Sir Charles Bell in his work on the Expression of the Emotions draws attention to the influence which powerful emotions exercise upon the respiration. MacMahon (1917) emphasizes that correct breathing has a calming effect on the emotions and has a good deal to do with recovery.

[2] Burns: Introduction to Biophysics, p. 243.

increased vagus activity or excitability. As a result there is a marked interference with the return flow of blood to the right side of the heart and venous distention occurs, with slowing of the cerebral circulation and an increase in cerebral venous pressure.

Mosso in his admirable monograph[1] has shown by observations upon men with trephined holes in the skull that intellectual activity was accompanied by an increased blood supply to the brain. He also showed by means of his plethysmographic studies that during intellectual activity the volumes of the arms diminishes with a great increase in the amount of blood to the brain. He demonstrated the latter by placing his subjects in a horizontal position on a delicately balanced table, so delicate was this balance that the table would vacillate with the subject's breathing. Invariably the head end of the table would descend whenever there was any intellectual activity on the part of the subject.

In another experiment performed on three trephined subjects, Mosso[1] recorded the brain pulse directly by tracings. He found that the slighest intellectual activity was accompanied by an increase in the blood supply to the brain. Bluemel (1913) quotes Mosso as follows: "One day, while experimenting with his female subject he noticed a sudden strengthening of the brain pulse and an increase in the volume of the brain for which he could detect no reason. Upon questioning the subject he ascertained that she had caught sight of a skull on the top of a cupboard, and that this had induced a slight fear in reminding her of her illness." With regard to emotion Mosso says in his classical work—"However, the changes in cerebral circulation produced by fear are far greater. The reprimands and threats that I cast at Bertino (the subject) when by moving his head or hands he interfered with the experiment and the disagreeable things I occasionally said with intention invariably produced a vigorous brain pulse. The pulsations became six or seven times greater than before, the bloodvessels expanded, and the brain swelled and beat with such vigor that my colleagues stared with

[1] La Paura (1884) which was translated both into German "Die Furcht" and into English "Fear."

astonishment at the photogram of the tracings." From these facts Bluemel (1913) states "it should be evident that there is really nothing remarkable in the ability of the stammerer or a mildly aphasic patient to speak when alone, though he may be unable to speak in the presence of other people. The mere presence of another person and the greater effort of attention required for conversation, causes an increased flow of blood to the brain and this on account of existing abnormal cerebral conditions, induces auditory amnesia. The mental imagery is most likely to be obscured when the subject is affected by intense emotion. Under these conditions cerebral congestion is greatest."

Robbins (1920) has extended Mosso's findings by a plethysmographic study of a trephined stammerer. He has shown that the act of stammering was accompanied by a "much more marked increase in brain volume than could be accounted for by either the physical or mental work used in normal speech." Whether this increased volume due to cerebral congestion is a causative factor or whether it is a result of the act of stammering is problematic. Of interest in this connection is the fact noted by Robbins that an increase in brain volume occurred fifteen seconds before speech began. It is, of course, possible that this may be purely psychic or mental phenomenon. However, there are certain facts that suggest other causative mechanism.

Travis (1927) in a photographic study of the voice in stammerers observed periodic fluctuations of breath pressure before tone was produced and short periods of vibration before the voice waves proper began. Froeschels (1916) found that in such cases there occurs abnormal oscillation of air in the nasal chambers and a disturbance of respiratory movements indicative of faulty diaphragmatic action. Other observers have mentioned this abnormality in the movements of the diaphragm and in a few of my cases I observed this phenomenon directly by means of the fluoroscope.

These observations, together with those reported in the present study, suggest that in stammerers there is a fundamental disturbance of the respiratory mechanism, probably of nervous origin, which results in an alteration in cerebral circulation leading to congestion before speech is actually begun. The respiratory

center is particularly sensitive to changes in circulation and to variations in the composition of the blood. Thus, superimposed upon an existing respiratory disfunction of nervous origin, there is a periodic respiratory abnormality of chemical origin due to changes in the pressure of oxygen and carbon dioxide in the blood supplying the higher cerebral centers. It therefore seems reasonable to conclude that one of the most prominent features of, if not a primary factor in the production of stammering is the disturbance of the mechanism of respiration outlined above.

SUMMARY AND CONCLUSIONS.

A recent reviewer of a book on "The Cause and Cure of Speech Disorders" has stated,[1] "The author who attempts to explain, or to treat stammering without attempting courageously to face the extraordinary and as yet unexplained phenomena of the speech disability itself—a disability that is predominantly responsible for the later severe emotional manifestations of the sufferer, is evading an as yet unsolved psycho-physiologic myster." In this thesis I have attempted to face these phenomena as they were exhibited by the 101 cases of stammering which I examined. I am convinced that multiple tests rather than dependence on a single method promise to yield information of the greatest significance in the study of stammerers.

The data, as I compiled them, at first suggested and gradually demonstrated the existence of a correlation between the respiratory and hematologic functions. The correlation resolved itself into a principle of physiologic compensation by which the subjects made up for the shallow inadequate breathing, characteristic of one-third of the adult cases, by increasing the red cells, red cell volume and the hemoglobin content of the blood. The compensation principle also took the form of periodic deep breaths in an attempt to make up for the mild anoxemia. While in those cases with normal respiration the hematologic findings were likewise normal.

The adult male group of 17 cases averaged as follows:

Volume per breath	426 cc.	Nineteen years	142 pounds
Minute ventilation	6.268 liters	Hemoglobin	95 per cent[2]
Respiratory rate	15	Red cell volume	49 per cent
Dilution factor	24	Red cells	6,224,000

In contrast, the adult male group of 13 cases averaged as follows:

Volume per breath	508 cc.	Nineteen years	135 pounds
Minute ventilation	7.084 liters	Hemoglobin	87 per cent
Respiratory rate	15	Red cell volume	42.8 per cent
Dilution factor	32	Red cells	4,667,000

[1] Jour. Am. Med. Assn., 1927, **89**, 1539.

[2] 100 per cent is equivalent to 16.25 gm. of hemoglobin.

While the adult group of 46 cases which stands midway between the two previous groups in hemato-respiratory data averaged as follows:

Volume per breath	455 cc.	Twenty and a half years,	136 pounds
Minute ventilation	6.750 liters	Hemoglobin	90 per cent
Respiratory rate	15½	Red cell volume	44.8 per cent
Dilution factor	28	Red cells	5,466,000

In respect to vital capacity the cases revealed the following data: Those having *high* vital capacity showed the highest frequency of shallow breathing, namely 50 per cent. The group having *normal* vital capacity showed a frequency of shallow breathing of 46 per cent. The group having vital capacity below normal showed the lowest frequency of shallow breathing, namely, 15 per cent. Low vital capacity was compensated for by breathing of a depth inversely proportional to the degree to which the vital capacity was below normal.

Finally, the hematologic compensation concomitant with rapid shallow breathing supports Bluemel's theory of cerebral congestion with resulting transitory auditory amnesia as the explanation of stammering.

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